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The Bancroft Memorial Lecture.[1]

DISORDERS OF THE PITUITARY BODY AND PARA-PITUITARY REGION.

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BEFORE I commence my address on "Disorders of the Pituitary Body" I desire to say that I deeply appreciate the honour you have done me in asking me to deliver this year the Bancroft Oration. I look upon your invitation as a generous-minded recognition of my long and sustained interest in the problems of clinical medicine and in the work of clinical teaching. It is, I feel, opportune to tell you that among the influences which stimulated my interest in clinical problems, even in my student days, was Dr. Joseph Bancroft's work on filaria in man. Over forty years ago, the late Sir Harry Allen, then Professor of Pathology and Anatomy at the University of Melbourne, told us of Bancroft's discovery of the filaria parent worm and told us that this world-famous investigation had been made by a busy medical practitioner in Queensland in 1877, and he suggested to us that if such a piece of original work was possible to Dr. Bancroft, then original and important observations should be possible to all of us in our medical work. As a matter of experience, original work in ordinary medical practice is possible to only very gifted persons, but keenness of interest in our ordinary work and alertness to keep ourselves well informed of all advances in medical science should be expected from everyone. You will realize that Dr. Bancroft's discovery was not the outcome of a chance observation, it was

[1] Delivered at Brisbane on May 3, 1929.

the outcome of live interest in the clinical problems of filariasis and it was further the outcome of his knowledge of the recent literature on the subject.

The Bancroft Oration which your immediate predecessors arranged should be delivered each year, commemorates not only Bancroft's original work, but also serves to recall his name for us as an example of that keenness and mental alertness which for thirty years he showed in his daily work here in Brisbane. In connexion with the personality of the man, all of you should read the delightful account of Dr. Joseph Bancroft written by Dr. E. Sandford Jackson and published in The Medical Journal of Australia of March 17, 1923.

I have chosen for the subject of this lecture "Disorders of the Pituitary Body and of the Para-Pituitary Region." These two structures constitute the pituitary apparatus as it is now called. The term para-pituitary is, of course, a hybrid word, but is more common than para-hypophysis and more convenient and obvious as a link with the terms hyper- and hypo-pituitarism: the para-pituitary region is to be taken as almost synonymous with the hypothalamic region of the brain.

I have chosen this subject, first, because I feel that with the recent advance in knowledge of these structures the subject must make a very special appeal to a large number of medical workers, to the physician and neurologist, to the pædiatrist and ophthalmologist to some extent, to the rhinologist in an important way, to the surgeon and to the radiologist, and, of course, to all science workers on biological problems. Secondly, I feel it to be the kind of subject that would have made a special appeal to the scientific-minded man in whose honour and memory the lectureship was established.

As an introduction to the clinical problems presented by the subject and in order that the structures which are to be discussed, may be clearly recognized, it is advisable to do a little light spade work and turn over, as it were, the ground which had been dug in the past in our more specially student days, when studying the anatomy of the cranium and its contents. Further, some knowledge of the development and embryonic basis of the pituitary body is essential to the understanding of certain clinical disorders of and about the pituitary apparatus.[1]

In any study of the topographical anatomy of the pituitary body it is from a surgical point of view important to realize that the part of that body situated in the *sella turcica* lies between two layers of the *dura mater*, the "periosteal" layer adheres to the floor of the sella and the "visceral" layer stretches as a diaphragm between the anterior and posterior clinoid processes, thereby practically shutting off the intrasellar contents from the intracranial structures. In all cases, with rare exceptions, an adenoma of the pituitary body is in its early stage a definitely intrasellar growth and consequently is a definitely extradural or extrameningeal growth. This anatomical position allows the surgeon and rhinologist to approach the tumour from below, for the purpose of partial removal or decompression, through the sphenoidal sinus, without fear of meningeal infection.

The pituitary apparatus is thrown out of gear obviously by an intrasellar growth and less obviously by a suprasellar growth and the position and nature of the growth must be, if possible, determined in the preoperative stage. In clinical work, when any pituitary disorder is even suspected, the determination of the size and contour of the *sella turcica* is, of course, of paramount importance; for this determination responsibility is placed on the radiologist and his interpretation of the radiographic film. It may here be stated that the size of the intrasellar space shows considerable variation in different normal individuals and it must be emphasized that slight, but it may be important, variations from the normal present great difficulties of interpretation. It is usually held that change in contour of the sellar space is more important than mere change in size, that the "balloon" contour is indicative of intrasellar pressure and that a flattening of the sellar space is indicative of suprasellar or downward pressure, whether from a growth or a general increased intracranial pressure. In any radiological interpretations of this part, it is wise to note the appearance of the bony base of the sella as well as the appearance of the bony clinoid processes. The degree of thickness of the bone separating the sella from the sphenoidal sinus and the degree of pneumatization of that sinus should be noted in all cases. Apart from the appearance of size and contour of the sella, any suspicion of abnormal radiographic shadows requires the most careful scrutiny because any even indefinite spots of calcification in or immediately above this space indicate the probable presence of a cranio-pharyngeal pouch tumour or possibly of a meningioma arising from the *tuberculum sellæ*, the olivary prominence of the older anatomists. The mention of a cranio-pharyngeal pouch tumour as a possible and a not very infrequent factor in producing disorder of the pituitary apparatus makes it necessary for me to remind you that from an embryological point of view the pituitary body must be considered as formed from a fusion of an upgrowth from the ectoderm of the primitive stomodæum with a process (infundibular) of the interbrain or diencephalon. This double development is a striking and interesting example of the "call of cell to cell" or cytoclesis, as it is termed by Professor Wood Jones in one of his fascinating books, "The Matrix of Mind." Now it has been shown that the intraglandular cleft between

[1] At this early stage in the address the topographical anatomy of the pituitary body and its relationship to the hypothalamic area of the brain were fully demonstrated by lantern slide illustrations, taken from diagrams, to be found in well known text books. The exact relationship between the pituitary body, the *tuber cinereum* and the mamillary body was demonstrated by means of illustrations taken from Dr. Philip Cloake's important paper on "The Influence of the Diencephalon on Metabolism," read before the Section of Neurology of the Royal Society of Medicine, April 28, 1927. Illustrations of the development of the pituitary body and of Rathke's cranio-pharyngeal pouch were taken from text books and from the article on "The Pituitary Adamantinomata," by Critchley and Ironside, in *Brain*, December, 1926.

the anterior lobe and the intermediate part of the pituitary body represents the primitive cavity of the cranio-pharyngeal or Rathke's pouch and that here or above the diaphragm of the sella in the infundibular stalk there are "epithelial rests" or ectoderm remains, like the cells of the embryonic enamel organ, from which develop these tumours, adamantinomata, as they have been called by English authorities. The significance attached to the word adamantinoma (to quote from Critchley and Ironside) "as used in the sense of pituitary neoplasm does not refer to the consistency of tumours in that region, but refers to their similarity in structure to the mandibular adamantinomata." Apart from this histopathological significance adamantinoma does not seem suitable as a clinical label for this type of tumour and instead I would commend the term craniopharyngioma (Cushing).

It seems to me that even a short clinical review of the problems of disorders of the pituitary apparatus would not be complete without reference to some remarkably good work that Dr. G. R. Cameron, a Melbourne University graduate, has recently carried out in Aschoff's laboratory at Freiburg, on the relationship of the *pars tuberalis* to the pituitary apparatus. He has sent me a copy of his work and I venture to reproduce one of the diagrams which he has published. It shows very clearly the relationship and extent of the various parts of the pituitary apparatus, and it definitely indicates that the intrasellar pituitary gland is only part of the apparatus which must be taken as also including the stalk (infundibular) as a partially glandular structure and as including the *tuber cinereum* and the mamillary bodies as intimately linked up with the *pars nervosa* of the pituitary body. The pituitary

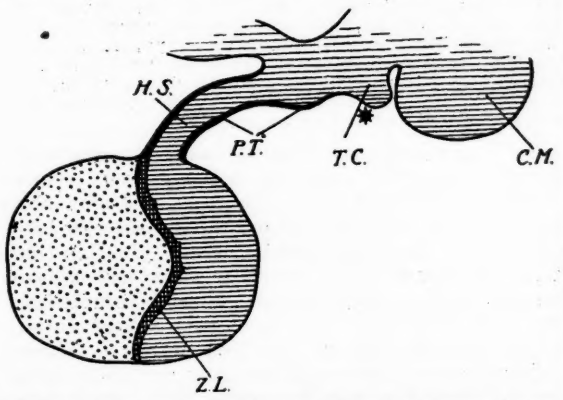

Diagram of the *pars intermedia-pars tuberalis* apparatus in man, showing the connexions of the *pars tuberalis* to the *pars nervosa*, to the infundibular stalk and to the *tuber cinereum*. The anterior lobe is shown by dots, the *pars intermedia* is cross-hatched, and the *pars nervosa*, the hypophyseal stalk, the *pars cinereum* and *corpus mammillare* are marked with straight horizontal lines. The *pars tuberalis* is black. C.M. = *corpus mammillare*. H.S. = hypophyseal stalk (infundibulum). (After Cameron.)

apparatus therefore is a comparatively extensive structure extending from the intrasellar lobes up to the hypothalamus or para-pituitary space. Seeing that this address is of the nature of a clinical lecture, I cannot allow myself to speculate on the phylogenetic significance of the hypothalamus and you must allow me to let inferences concerning the physiology of the pituitary apparatus emerge as certain comparatively recent experimental work is reviewed and certain types of clinical cases are discussed.

At this point I desire to say frankly that this address on this subject would not have been possible for me unless I had followed for many years Cushing's work on the problems of pituitary disorders and unless I had read and studied his remarkably illuminating lecture on "Acromegaly from a Surgical Standpoint," delivered as the annual oration before the Medical Society of London in June, 1927. In a period of two years, from 1925 when Cushing dealt with some of these problems in the Cameron Lectures, delivered in Edinburgh, to the middle of 1927, an immense advance, not only in experimental methods had been made by Professor H. M. Evans and Professor P. E. Smith, but also in clinical observations, great advance had been made by Cushing himself. It seems to me that the whole world must gratefully acknowledge its indebtedness to Cushing in all that appertains to pituitary disorders and intracranial surgery. Any value that may be attached to this address will be largely lost unless you read in connexion with it Cushing's lecture on the subject in *The British Medical Journal* of July 2 and 9, 1927.

In the elucidation of the problems of pituitary disorder clinical observations and experimental work have gone hand in hand and now for the last ten to twenty years there has been accumulating a body of evidence that certain defects and change in the pituitary gland were associated with defect of growth, such as dwarfism and other bodily defects; and there was the converse clinical evidence, long known, that associated with certain pituitary tumours there was overgrowth of the skeletal tissues and bodily organs, producing acromegaly or gigantism. Except for some suggestive work by the late Brailsford Robertson, until his recent death Professor of Physiology in the University of Adelaide, and some even more suggestive work by Professor P. E. Smith, of the Leland Stanford University, no convincing evidence of experimentally produced overgrowth was forthcoming. On the whole, it may be said that until recently clinical and pathological observations were flounderingly in advance of experimental work.

Recently, however, Professor Evans, of the University of California, found that by the repeated intraperitoneal injections of a fresh extract of the anterior lobe of the bovine pituitary gland overgrowth of the body to the degree of gigantism with hyperplasia of thyreoid, parathyreoid and of the adrenal cortex could be produced in rats. The same effect could be produced by repeated homoplastic implantation of the anterior lobe. These observations have been convincingly confirmed as correct by other workers. It is to be noted that Professor Evans also proved that the injection of this fresh

extract would stop the œstral cycle in adult female rats and would prevent its occurrence in preadolescent animals. It is curious to observe in this connexion that controlled experiments show that both excess of anterior lobe extract and defect of anterior lobe secretion are associated with defect of sexual activity; this experimental evidence confirms clinical observation in this respect in cases of hyper- and hypopituitarism. It is perhaps wise to say that the injections of "commercial" anterior lobe extract were proved to be without effect and that at present no reliable form of this extract is yet available for clinical work, though undoubtedly that will come, as "Insulin" and thyroxin have come.

In contrast to this experimental production of gigantism, Professor P. E. Smith, of the Leland Stanford University, has carried out a series of experiments by which, following the surgical destruction of the anterior lobe of the pituitary body, he has produced dwarfism in rats. With arrest of growth there has also been arrest of sexual function with atrophy of the thyreoid gland and adrenal cortex, but no adiposity was produced. We see, therefore, in this particular series of experiments the production of a condition counterpart to the clinical condition of dwarfism of the Symonds or Lorain type.

In relation to these striking observations a third set of experimental investigations must now be described. Professor Smith in February, 1924, by an ingeniously devised and skilful operation injured the hypothalamic area (the para-pituitary area) without extirpation of any part of the intrasellar portion of the pituitary apparatus. What happened? A year later the experimental animal, a female rat, had grown very fat, it weighed 840 grammes, as compared with its litter-mate sister weighing 299 grammes; and when it was killed "extensive atrophy of the sex apparatus was disclosed, the other ductless glands being of normal size." It will be seen that this third type of experiment produced a condition which strongly suggests comparison with the clinical condition of adiposo-genital dystrophy (Fröhlich).[1]

Before I pass on to consider the classification and some of the signs and symptoms of clinical cases of disorders of the pituitary apparatus, disorders which have been so strikingly elucidated by the experiments quoted, I may perhaps call your attention, first, to the negative part that the posterior lobe plays in these experiments and, secondly, to the unrecognized function of this lobe in any human disease, except, possibly, *diabetes insipidus*.

Yet from the so-called posterior lobe which, of course, experimentally at least, must be difficult to separate from the *pars intermedia* and portion of the *pars tuberatis*, there have been obtained extracts which have well known and striking pharmacological actions. As Dott points out, we have as yet "no definite information as to the function of the posterior lobe in mammals. It is quite unwarranted by known facts to attribute any definite clinical symptom to a lesion of this lobe. We must note, however, with great interest the effects of its extract in enhancing para-sympathetic activities and in antagonizing adrenalin and sympathetic activities." From a clinical point of view, therefore, we must leave in doubt the function and significance of the posterior lobe or *pars nervosa*, being prepared, as is reasonable, to link it up with some of the obscure functions at present attributed to disturbance of the hypothalamic region of the brain. This view is suggested by Dr. Cameron's diagram.

In any clinical classification of pituitary disorders it is helpful to divide cases broadly into two classes: (i) Those indicating hypopituitarism and (ii) those with hyperpituitarism. It is further necessary to make a subdivision of these two broad classes, according to the age incidence of the onset of the disorder, for example, gigantism and acromegaly. And, third, it is necessary to determine broadly the pathological basis of the disorder, whether the syndrome is secondary to a tumour growth or whether the symptoms are primarily due to some inflammatory or vascular change about the pituitary apparatus. Let me quote from Cushing's lecture a case of hypopituitarism (anterior lobe defect) coming on in a child and not associated with a tumour. Those of you who have had opportunities of working in a children's hospital, will recall many similar cases, but it is helpful to have Cushing's authority behind us.[1]

The patient was a girl, aged twenty-eight years, active and intelligent, but a Lilliputian dwarf, with the physical development of a child of eleven years old. Though she had the figure of a child, she had the face of a middle-aged woman. She had never menstruated. She had no defect of fields of vision. On X ray examination the *sella turcica* was of small size, but of normal contour, the cranial bones were thin and the epiphyseal lines of the long bones were present. The past history of this case was that the patient was a normal child until she got a septic throat when eight years old; since then she had never been well and had grown very little.

Cushing regards the condition as due to defect of anterior lobe, the result of a non-virulent septic embolic infection of some of the vessels of that lobe, producing an ischæmic necrosis of the anterior lobe and constituting Simmonds's disease. To this condition of infantilism or dwarfism many names have been given, Simmonds's disease, Lorain's infantilism, ateleiosis, or if "senilism" is associated with infantilism, the term progeria has been suggested. These children do not grow up, but they grow old; they are all probably manifestations of anterior lobe deficiency of some obscure origin.

I have seen one case which I have recognized, of a similar character coming on in a middle-aged woman after some puerperal infection. Many years ago I regarded it as an obscure and atypical form of myxœdema with atrophy of the skin and cessation of menstruation; more recently the basal metabolic rate was found to be low and all along she has responded slightly to the use of thyreoid gland extract.

[1] Lantern slide illustrations of the three conditions—gigantism, dwarfism and adiposity—in experimental rats with controls of normal litter-mate rats were shown.

[1] Lantern slide illustrations of all clinical types of pituitary disorder were shown.

To pass on to a similar type of case but with a very dissimilar primary cause, let me quote the following case.

Here, too, the patient was a very undersized child who had, unlike the previous patient, suffered from headache and vomiting with some polyuria, coming on gradually for about four months. On examination pallor of the optic discs was present with bitemporal hemianopia and radiographically there was found a widened *sella turcica* with a ring of calcareous nodules within the sella and more calcareous nodules in the suprasellar region. The X ray appearances and the primary optic atrophy and the clinical history combined to show that the condition of defective anterior lobe function was secondary to a partially calcified cystic growth, a cranio-pharyngioma.[1] In Cushing's hands this centrally situated growth was successfully removed by "the transfrontal approach." About two years later the child had regained her full fields of vision, had lost the polyuria, had increased in weight and had grown slightly.

It is clear that the only hope for these cases of cranio-pharyngioma, as with meningioma growing from the tuberculum of the sella, is by operation, and yet it must be recognized that no intracranial operation requires more skill and more knowledge. Until recently the experience of English surgeons of great repute has been gravely disappointing, both in these cases of cranio-pharyngioma and also in cases of suprasellar meningioma. I know of a case of a suprasellar pituitary tumour successfully operated on by Sir Henry Newland, of Adelaide, by the transfrontal approach, and encouraging reports are recorded by Mr. Norman Dott and Mr. Hugh Cairns who, it may interest you to know, was in his undergraduate days a student at the Adelaide Medical School.

In this connexion, may I commend to surgeons, ophthalmologists and physicians the intensely interesting articles on meningioma arising from the *tuberculum sellæ*, by Cushing and Eisenhardt, published in *The Archives of Ophthalmology*, January and February of this year, 1929.

Before I leave this part of my subject I desire to say that it is only within the last three years that I had any real understanding of the syndrome produced by a cranio-pharyngioma and such knowledge as I then obtained was due to the article by Critchley and Ironside which appeared in *Brain* in 1926. I then realized that I had misinterpreted the significance of several cases and that among other matters I had not realized that these congenital tumours may be without appreciable symptoms until adult life, then producing the syndrome of signs and symptoms which indicate pressure on the optic chiasma, pressure upwards on the hypothalamic area and pressure downwards on the anterior lobe.

Leaving for a few minutes the consideration of defects of the pituitary apparatus associated with tumour growth, I desire to remind you of one clinical type of disturbance apparently associated with changes in the hypothalamic or para-pituitary part of that apparatus. You will remember the rat experiments, how defect of the anterior lobe was shown to produce dwarfism and how hypothalamic injury was followed by adiposity and genital atrophy. Similarly, you will know, we have clinical examples of adiposo-genital dystrophy, the so-called and well known Fröhlich's syndrome, in growing children. In many of these cases there is no sort of evidence of any tumour growth and the condition may be due to some chronic inflammatory or vascular changes in the para-pituitary space. Besides fatness and genital atrophy, there may be obscure disturbances, such as high sugar tolerance, *diabetes insipidus*, undue somnolence and other bizarre symptoms which still require much elucidation. As a practical clinical point it seems to me that every case of Fröhlich's syndrome in the child, or even of that rare condition in the adult, *adiposis dolorosa*, should be searchingly and repeatedly investigated instead of just being treated by thyreoid extract.

I now propose to leave the suprasellar area and pass on to the consideration of adenomatous tumours growing in the intrasellar space. Pituitary adenomata are of two kinds, classified according to their cell-staining reaction; the cells of one, the commoner of the two types, do not stain with eosin, the cells of the other type have a special affinity for eosin. Consequently the first type is labelled a "chromophobe" adenoma and the second is classified as a "chromophile" adenoma. It is apparently a fact, and very remarkable, that in spite of identity of situation and similarity in size and structure, these two types of tumour produce very different effects. Associated with the chromophobe adenoma there is produced a hypopituitary state, while the chromophile adenoma produces hyperpituitary manifestations with overgrowth of bodily tissues, as is seen in acromegaly. The clinical diagnosis of a minor degree of hypopituitarism associated with a chromophobe adenoma is very difficult and cannot be made convincingly without the presence of signs of local pressure. Cushing believes that the disorder is a very common one, with indefinite manifestations of diminished function of the anterior lobe, such as loss of hair, atrophy of skin, lowered basal metabolism and a tendency to adiposity and sexual dystrophy. These symptoms are common enough in any elderly person and are frequently regarded as of a myxœdematous nature. Seeing that we have at present no medicinal remedy for anterior lobe defect, it may be held that no great harm follows from this error in diagnosis, but it must be recognized that the prognosis is entirely different in myxœdema and this form of hypopituitarism. I have recently seen in consultation such a case, an anomalous type of myxœdema, treated with thyreoid extract; then the patient became profoundly asthenic with a certain amount of wasting. She died and nothing abnormal was found at the autopsy except a pituitary adenoma. Beware, then, of anomalous cases of myxœdema. When, however, the chromophobe adenoma enlarges, definite pressure on the optic chiasma will begin to be manifest. Some defect of vision is noticed by the patient and unless the fields of vision are most carefully determined

[1] The radiologist will know that different degrees of penetration are sometimes required to make manifest in radiograms small and indefinite areas of calcification in this situation.

by the oculist, the condition is overlooked or mistaken for a condition of retrobulbar neuritis. Even before chiasmal signs of slight or gross bitemporal hemianopia are present, the X ray examination may reveal a slightly "ballooned" sella. This change, as I have pointed out, is difficult to determine, but help may be obtained by careful attention to the condition of thinness of the bony floor of the space.

When pressure signs are present, surgical decompression of the sella by the transphenoidal route is at present the chief method of treatment. Following the operation deep X ray therapy is of apparent value. Probably the implantation of radon seeds may prove to be of special value in preventing recurrence of the adenoma. I need hardly point out that the satisfactory investigation and treatment of this form of hypopituitarism mean close cooperation between physician, oculist, radiologist and specialist surgeon.

There remains now for me to refer briefly to the comparatively rare types of cases, known as gigantism and acromegaly; both of them are associated with the presence of a pituitary chromophile adenoma. In gigantism the adenoma has occurred in the preadolescent period of life (an exceptionally rare occurrence), while acromegaly is associated with the occurrence of the adenoma in adult life.

In connexion with the practical clinical problems of these cases and avoiding text book statements, I wish to refer to two matters of importance. First, the surgical operation by the transphenoidal route is free from appreciable danger, and the operation is followed by subsidence of the signs and symptoms of the disorder. I lay some emphasis on this matter because recently I have seen a case in consultation.

A lady had marked overt signs and obvious symptoms of acromegaly. She refused operation because she was advised by another physician that it was a most dangerous and difficult operation (and so it is, by the transfrontal route) and because she was advised that beyond problematical and temporary relief of her headache and vision, the operation would do no good.

Second, I would call your attention to the symptoms of hyperpituitarism before the acromegalic signs are obviously present. These symptoms are muscular and general weakness, with increase in size of body tissues. Glycosuria or a lessened sugar tolerance, an increased metabolic rate and genital dystrophy—as you will see, a heterogeneous mass of symptoms, until recently very difficult to understand and even now not clearly to be explained. I have such a case under observation now.

In illustration of these two sets of clinical problems let me quote again one of Cushing's cases.

A woman, aged twenty-two years, was seen in December, 1925. She complained of troublesome headache for two years. She stated that she had been married at nineteen years of age; a healthy child was born to her a year later, but headaches began during her pregnancy. Menstruation recurred after pregnancy in the normal way, but she noticed then that her features had become coarse, her hands and feet "puffy," her skin hirsute and her weight had increased. On examination she had a palpable thyreoid gland and a basal metabolism of +25%. There was slight glycosuria. Radiography revealed no definite enlargement of the sella. Eight months later the headaches were so bad and continuous as to be completely incapacitating, the basal metabolism was still +25%, and glycosuria was still present. After some observation and ineffective treatment it was decided that hyperpituitarism could alone account for her symptoms. Accordingly, by the customary transphenoidal route, a fairly radical extirpation of the sellar content was made; the tissue was typical of a chromophile adenoma. After the operation the headaches entirely ceased, glycosuria disappeared, the basal metabolism gradually dropped to +3% and definite diminution in body weight and in size of hands and feet occurred.

With the citation of this instructive case I close this clinical lecture on disorders of the pituitary apparatus. I trust that from the mass of material I have presented something may have emerged that may be of occasional help to you in your work; and perhaps I may venture to hope that by this oration I have in some minor degree stimulated your interests in these clinical problems in the way that Dr. Joseph Bancroft would have wished.

DISEASE OF THE GALL BLADDER.[1]

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The most difficult problems confronting the surgeon in gall bladder disease are making a correct diagnosis and then, having decided that the gall bladder is the offending organ, what treatment should be adopted.

The hour that has been allotted to me, will permit of little more than a skirmish around the subject, and my remarks will be directed more particularly to the work carried out in many parts of the world seeking the cause of gall bladder infection which Wilkie[1] considers the commonest of all abdominal maladies. As a prologue it might interest you to hear a short historical sketch, but for a fuller dissertation Stewart McKay's[2] history of Lawson Tait and Moynihan's[3] book on gall stones and their surgical treatment should be consulted.

It is only two weeks ago that a decision was made to speak of one particular subject instead of demonstrating surgical cases, compelling me to draw upon material that has appeared in a previous paper.[4]

Alkmaion,[5] of Crotona, about 500 B.C., was the first to introduce abdominal section, and thus must be considered the pioneer in paving the way for the investigation of the pathology of abdominal conditions. His pupil, Philolaos,[6] propounded the theory that disease was due to four elements—black bile, yellow bile, blood, phlegm. History also tells us that the term black bile was known to Aristophanes.

Alexander Traillanus,[7] about A.D. 600, was the first to mention the occurrence of gall stones, but Antonio Benevieni[8] who practised in Florence in the last third of the fifteenth century, was the first

[1] A post-graduate lecture delivered at Sydney on June 6, 1930.

in modern times to conduct *post mortem* examinations, and amongst his findings gall stones are mentioned. Fabricius Hildanus,[9] in 1618, was the first to remove a gall stone from the gall bladder of a living patient.

Petit[10] (1674 to 1750), a French surgeon, founded the surgery of the gall bladder, but Bobbs,[11] of Indianapolis, was the first to perform the modern operation of cholecystotomy.

Three cases of gall stones operated upon by Keen[12] (1879), Sims[13] (1879), and Tait[14] (1879) cleared the ground for the modern operations practised on the gall bladder today. Sims it was who coined the name cholecystotomy, while Tait was the first to carry out cholecystotomy in England successfully, although he had removed stones from a suppurating sinus in 1869—ten years previous to the above operation—and Ochsner[15] gives him the credit for placing the surgery of the liver and gall bladder on a firm basis. His pupil, Mayo Robson, worthily carried on his work and handed down his genius to his student, Moynihan.

To America we owe much, particularly to Murphy, the Mayos and their wonderful array of scientific investigators, and to Graham. But to Langenbuch,[16] of Germany (1882), honour is due for the first attempt made to remove the gall bladder (cholecystectomy) and to the same surgeon two years later for performing choledochotomy to dislodge a stone from the common bile duct, though Knowsley Thornton[17] was the first surgeon to remove successfully a stone from the common duct.

Germany still leads with the first operation of cholecystenterostomy which was performed by von Winwarten[18] in 1882.

Duodeno-choledochotomy, that is, transduodenal attack on the common bile duct which Moynihan thinks highly of as a means of attacking common duct stone, was first performed in 1884 by McBurney[19] and in the same year by Pozzi[20] and Kocher.[21]

Choledocho-duodenostomy, a popular method in use to overcome stricture of the common bile duct, was first performed by Riedel[22] unsuccessfully in 1884, but by Sprengel[23] successfully in the same year. Why this operation was attempted so early in the history of gall bladder surgery I have not been able to discover, but apparently, like most of us, they found injury to the common bile duct a comparatively common occurrence.

Thus we learn that gall bladder surgery, as we know it today, is a development of quite recent times, and still much research is going on, leaving in its wake a trail of uncertainty.

Development.

The gall bladder, being an outgrowth from the common bile duct, which is developed from the primitive foregut, has an epithelial lining and a mesodermic background and is subject to all forms of growths, as any other similarly constituted structure of the human body is; for the growths outside of carcinoma are extremely rare indeed and negligible; but of carcinoma just a few remarks.

Almost invariably in carcinoma of the gall bladder a previous history of gall bladder inflammation is found and at operation in most cases calculi are met. Archibald Leitch[24] describes many interesting experiments on guinea-pigs, in which he was able to produce carcinoma. He introduced into the gall bladder, in some pebbles, in others gall stones, showing that the chemical composition of the calculus plays no part whatever, but that the carcinoma is the result of mechanical stimulation. His contention is that the constant mechanical irritation of calculi, operating on a suitable tissue by itself, can produce carcinoma, and that in those cases of carcinoma of the gall bladder, where no stones are found, the growth is due to some other causal agent or agents not identified. These views are confirmed in practice, it being usual to find in malignant cases of gall bladder a long history of cholecystitis preceding the symptoms of the malignant change, which is indeed a strong argument for operation.

Function.

Now let us consider the function of the gall bladder.

The gall bladder is not found universally amongst animals. It is absent in the horse, ass, zebra, quagga, rat, pocket gopher, rhinoceros, tapir and pigeon. In the horse, it is of interest to note in the light of remarks to follow, a single choledochus opens into the duodenum by way of the ampulla of Vater.

All the cud chewers possess gall bladders, such as the ox, sheep, the camel *et cetera*, and so do pigs, dogs and cats.

1. By some its function is said to be that of a reservoir for the flooding of the duodenum by bile, when the food passes through this part of the intestinal canal, but this theory is hard to accept, as about 1·5 litres (fifty ounces) of bile are excreted in the human in twenty-four hours and the capacity of the gall bladder is only fifteen cubic centimetres (one ounce). As man feeds at from four to five hour intervals, the gall bladder can hardly be considered in man to have much functional value as a reservoir influencing the admixture of bile and food. And, too, if the gall bladder were a reservoir, an intrinsic mechanism would be expected, such as a strong musculature, but only a very weak one exists.

2. W. J. Mayo[25] speaks of the gall bladder as a safety valve for taking the tension from the common and hepatic ducts and secreting mucus, reducing thereby the risks of pancreatic and other complications; yet, in the horse, whose anatomy and pathology have been studied most minutely and which does not possess a gall bladder, a common opening exists at the papilla of Vater for the common bile and pancreatic ducts and pancreatitis is practically unknown. On the other hand, in the families of which the ox and pig are members, there are separate openings into the duodenum for

the common bile duct and pancreatic ducts, and these animals have gall bladders, therefore rending asunder the safety valve theory.

3. Some authorities consider the gall bladder an active concentrating chamber in which bile may be concentrated at least ten times in twenty-four hours by its active absorptive powers. It can be understood, with such absorptive powers, that when infection supervenes, the inspissation of bile taking place tends to calculus formation and that the rapid absorption of toxic products may cause considerable poisoning of other tissues, such as those of the heart. By experiments on dogs Rous and McMaster[26] proved this theory beyond all doubt. Harer, Hargis, Van Meter[27] further elaborated on this theory by a work of great merit and proved that the concentration was effected chiefly by the lymphatics which carried infection from the gall bladder to glands at the head of the pancreas, producing lymphangitis and lymph stasis, with subsequent organization of the inflammatory mass resulting in chronic pancreatitis. It is my experience that chronic pancreatitis is a very common condition, particularly associated with cholecystitis, which Rutherford Morison[28] pointed out as far back as 1922 as being associated with cholelithiasis.

4. Woods Hutchinson[29] and others consider the gall bladder a vestigial structure, but why, then, is it so well formed in man who stands highest in the scale of evolution, as it is in the lowest vertebrate?

5. Another function assigned to the gall bladder is that of a regulator of the flow of bile, but Rost's[30] experiments point to the flow of bile into the intestines being regulated by the muscle of Oddi rather than by the presence or absence of the gall bladder.

6. Still another function assigned to the gall bladder is that of an active secreting organ elaborating and adding to the bile something which is of importance, either to the general body economy or to the mechanism of bile expulsion or its chemical action. In this connexion it is of interest to note that cystic bile is ten times richer in solids than hepatic bile and contains considerably more mucin.

Anatomy.

Having considered the function of the gall bladder, let us now refresh our memories of the anatomy of the gall bladder and ducts, and for most of the remarks to follow I am indebted to a fellow student and friend, Wingate Todd.[31] The gall bladder lies on the visceral aspect of the liver to the right of the quadrate lobe and between the ventral border of the liver and its transverse fissure. Occasionally it is embedded in the substance of the organ and in rare cases it is absent. It is pyriform or cylindrical in shape, has a rounded extremity or fundus, a body or main part with upper and lower aspects, a neck continuous with the cystic duct. Its direction is from before backward, upward and to the left.

The fundus is from ten to twelve centimetres, but the neck only three and a half centimetres from the middle line and the latter, just above Addison's plane, that is, at the level of the first lumbar vertebra.

The body of the gall bladder is bound to the liver by intervening cellular tissue and through this pass the lesser cystic vessels. Sometimes the body of the gall bladder is suspended by a mesentery from the liver and in this type one finds a "flopping" gall bladder often the seat of biliary stasis and even simulating cholelithiasis.

The cysto-colic ligament, that is, the outlying portion of the lesser omentum, is present in one out of four individuals, and passes from the undersurface of the gall bladder to the second portion of the duodenum.

The neck of the gall bladder forms an acute angle in a direction parallel with the body. The body, the neck and the cystic duct form a Z-shaped structure, and at each bend there is a reduplication of the mucous membrane forming a valve-like partition which, with several other partial foldings of the mucus membrane of the gall bladder, make passage of a probe into the cystic duct almost impossible. Diverticula are frequent at the junction of the body and neck, and one in particular is known as Hartmann's pouch and is often the starting point of stones.

The cystic duct is the narrowest part of the bile passages and passes up and to the left and then turns down and meets the hepatic duct. The cystic and hepatic ducts lie in the right free margin of the lesser omentum in front of the foramen of Winslow and join behind the first part of the duodenum to form the common duct. It is when the cystic duct is dilated after a recent attack of biliary colic, even though there be stones in the gall bladder and no history of jaundice, that one must be on the alert for a pebble in the common duct.

The common bile duct which in rare cases is represented by two channels, consists of a retroduodenal, a pancreatic and an intramural portion. The duodenal part is in close relation with the inferior *vena cava*, the portal vein and gastroduodenal trunk of the hepatic artery, and according to Rost[32] it opens into the bowel by Vater's papilla in 78% by a single orifice and no fluid can be forced into the ducts by distending the duodenum. The common bile duct gets smaller in calibre as it reaches the duodenum and thus stones are more frequently met in this position.

The blood supply of the gall bladder comes from two sources, from the cystic artery itself and from the lesser cystic arteries from the liver. The cystic artery usually arises either from the hepatic trunk or from the right branch of the hepatic and lies above and to the left of the cystic duct.

The cystic duct gets its blood supply from the cystic artery, the hepatic ducts and upper part of the common bile duct receive their blood supply from the hepatic trunk, while the lower part of the common duct gets its blood supply from the right

superior pancreatic-duodenal artery. The habit of crushing the cystic duct before applying a ligature to it — and so distinguished an authority as Moynihan recommends it—is dangerous. The ligature is generally placed in the crushed area which must soon slough and is liable to cause leakage of bile. It is preferable to define clearly the cystic duct, apply the ligature and then clamp distally. In this way the blood supply of the stump is left intact, and natural and quick repair and closure of the cut end take place.

The portal vein lies to the lateral side of the upper third of the common duct and may overlap it in front, and is liable to injury in operations on this part of the common duct. As it is this part of the common duct which should be opened when exploring for stones, this relationship must ever be kept in mind. I have seen the portal vein opened more than once.

The lymphatics of the gall bladder go to the hilum of the liver and join the efferent vessels of the liver and connect with those from the common duct. They all unite in a plexus and with those of the duodenum drain into nodes in the immediate neighbourhood of the head of the pancreas.

Experiments of Berceanu[33] have proved the existence of a direct lymphatic path from the appendix to glands in the duodenum-pancreatic region, and it would thus seem that the lymphatics are the chief connecting links between an evil triumvirate of the abdomen—the appendix, the gall bladder and the pancreas. The close association of the duodenum with these structures has just been pointed out, and it can be readily understood how easily infection in any of them can set up trouble in this part of the digestive tract and, as mentioned later, often leaves as a legacy intractable perio-duodenitis.

The main nerve supply (Mackenzie[34]) to the gall bladder comes from the eighth and ninth dorsal nerves *viâ* the solar plexus and, according to Bainbridge and Dale's[35] experiments, the vagus nerve gives some motor fibres.

The phrenic nerve (third, fourth and fifth cervical nerves) is also considered to give a supply and by these paths referred pain from the gall bladder and ducts is frequently found in the right lower scapular region (eighth dorsal nerve) and in the right arm and right upper scapular region (fourth and fifth cervical nerves).

The names of different observers have been handed down in connexion with pain and tenderness over the gall bladder. The direct pain felt over the fundus of the gall bladder is known as Boas's sign, whereas Murphy's sign depends on local peritonitis and is elicited as tenderness on deep pressure under and just below the ninth right costal cartilage. Mayo Robson's spot is an area of tenderness on deep pressure at a point at the junction of the middle and inner thirds of a line joining the tip of the ninth right costal cartilage to the navel.

The Cause of Infection.

The slides shown you have demonstrated the close association of the gall bladder and ducts with neighbouring viscera of great importance and thus it will be readily understood with what ease infection from adjacent parts can pass to the gall bladder and *vice versa*. While reviewing gall bladder infections, calculus formation comes to mind — a condition due not only to infection, but to disordered metabolism as well. In many patients operated upon with a history of cholecystitis of long standing the gall bladder shows, both microscopically and macroscopically, signs of a long-standing infection and yet no trace of stones can be found nor a history of the passage of stones obtained, which points to there being a third causative factor, namely, a predisposition to stone formation.

Rolleston[36] lays special emphasis on the metabolic factor as a cause of stones. He asserts that this factor sometimes acts alone, as in cholesterol formation, at other times the infective factor alone is the cause, but more often the two work together.

The gall bladder is like any other organ of the human body in that infection may be derived from any source, such as infected teeth, nasal sinuses, alimentary tract, genito-urinary system, and when once infection enters the blood it can get access to any part of the body and to the gall bladder especially, if the liver has to act the part of the septic tank and cleanse the fluids passing through.

Infection may reach the gall bladder by many routes, from the duodenum, by way of the common duct—and it is of interest to note that Butt[37] reports a case of round worm in the gall bladder—from the liver through the portal circulation and thence by the bile, from the blood and lymphatics; or attack may come through adhesions of the gall bladder with some neighbouring organ. Much work has been carried out recently in achlorhydria, a very prevalent state, for Hurst[38] in 762 consecutive cases found achlorhydria in 15%, and Langdon Brown[39] states 5% of the population are achlorhydrics. In many of them a streptococcus has been found in the duodenum and in a series of gall bladder cases investigated for me by Dr. Inglis[40] and his assistants in the Pathological Department of Sydney Hospital, achlorhydria has been in the ascendant. Thus this condition of achlorhydria and its attendant duodenal infection must also be considered as a possible cause of infection of the gall bladder, but is it *propter* or *post hoc*?

When bile is the direct source of infection the mucosa is first attacked and the inflammation passes outward through the gall bladder wall to the liver and surrounding organs; whereas if the infection is blood borne, for example, in typhoid fever, the submucosa is first infected, when the spread is both inwards to the mucosa and outwards to the liver and peritoneum. If, on the other hand, invasion takes place through the lymph stream or from adherent organs, the serous coat is first affected.

The feeling amongst most investigators today is that infection of the gall bladder is mainly blood borne.

Research goes to show that gall stones, apart from the pure cholesterol stone, are preceded by an infection, and in support of this Gross[41] found in 9,531 autopsies 802 cases of gall stones. There was a definite association between faceted pigmented stones and cholecystitis, but no cholecystitis existing with cholesterol stones. Walton[42] lays stress on the connexion between urinary and gall bladder infections, pointing out that the two conditions are closely associated and are manifestations of chronic septicæmia. His opinion is that the *Bacillus coli communis* organism being more common in pregnant women would help explain as one factor the more frequent occurrence of gall stones in women than in men (two to one), though, of course, constipation is more prevalent in women. Out of 347 women patients of his in the London Hospital afflicted with gall stones, 324 had been pregnant.

Extensive and searching experiments by Rosenow[43] on animals by intravenous injections of bacteria with proven affinity for various abdominal organs, for example, stomach, gall bladder, duodenum and appendix, demonstrated that he was able in a great percentage of cases to produce inflammations in these organs typical of where they were cultured from, but he was unable to produce cholecystitis by injecting them directly into the gall bladder.

By experimenting with numerous varieties his results showed the streptococcus to be the chief cause of ulcer of the stomach, cholecystitis, appendicitis, pancreatitis, but when streptococci and *Bacillus coli communis* were together injected, cholecystitis became very marked. Moynihan's[43A] findings, on the contrary, in one hundred consecutive cases point to the *Bacillus coli communis* organism being the most frequent causative one, namely, in nearly 50%.

Gall bladder infection may be so severe as to cause perforation and sudden death of the individual and, even if this catastrophe does not happen, the condition will often pass to acute suppurative pancreatitis with almost certain fatality. Any acute infection of it, even if subsidence does occur, leaves behind a more or less crippled gall bladder, incapable of getting rid of its contents efficiently, either by the cystic duct or lymph stream, and thus a *cul-de-sac*, the seat of chronic sepsis, a centre for calculus formation or implantation of carcinoma, and from which toxins are absorbed, having deleterious effects locally and on the body mechanism as a whole, for example, the heart.

Observations on gall bladders removed by several authorities have demonstrated sterile bile in the gall bladder and in the same gall bladder wall numerous organisms have been shown; yet, from the same gall bladder no growth of organisms has been obtainable. Researches carried out by Dr. Inglis[40] and his coworkers for me show a very low percentage of organisms possible of culture in the inflamed gall bladder, which we hope to speak about at some later date. A. L. Wilkie,[44] too, got very low positive cultural results from the inflamed gall bladder, but very high from the cystic gland. Nickel and Judd[45] have found the majority of chronic inflamed gall bladders sterile and the same in the strawberry gall bladder. This observation is a very important one, demonstrating that, because no organismal growth can be obtained, the presence of active infection cannot be ruled out. A similar analogy is met in tubal infections, even excluding gonococcal infections, where active infection is obviously present, yet no growth can be obtained by culture.

To sum up the pathology, what happens in most gall bladder infections is that biliary stasis takes place, with probably an increased cholesterol content in the blood and bile associated with constipation, then infection of the portal blood resulting in cholecystitis. It must be remembered that biliary infection is not necessarily a precursor of stone formation, for example, cholesterol stone formation is usually aseptic, though even in these cases infection soon takes place.

The Symptoms.

The symptoms of gall bladder trouble are generally referred to the stomach. Most frequently there is a flatulent dyspepsia in the form of discomfort a half hour after food, relieved by the belching of wind and more pronounced after taking certain foods, such as fat, pastry and vegetables. In others the gastric symptoms show themselves an hour or more after food, as heart burn and acid eructations.

Pain is a very important symptom in all cases of cholecystitis. It may be no more than a little discomfort after food to that of severe pain referred to the right costal margin and round the lower right ribs to the lower angle of the scapula *viâ* the eighth dorsal nerve. In a small minority the pain extends to the right iliac fossa, simulating appendicitis or renal trouble and an important point to remember is that the three conditions may coexist.

On occasions pain on the left side is the more prominent and, when accompanied with palpitation, suggests to the patient cardiac trouble, a condition spoken of by physicians as "cholecystic heart." We have found almost invariably some degree of pancreatitis in these cases and presume the cardiac pain is transmitted probably through stomachic adhesions by means of the left vagus. In those cases where the pain is referred through the fourth and fifth cervical nerves, neuritis is suggested from the aching in the right shoulder, right side of the neck and down the right arm.

Occasionally symptoms are referred to the right breast *viâ* the eighth dorsal nerve, and it can be readily understood that when so isolated a structure as the gall bladder becomes diseased and unable normally to discharge its contents, absorption of the noxious accumulations must have dire effects on the general body mechanism, even to causing

muscular rheumatism and rheumatoid joints. Wilkie[46] mentions relief from cholecystectomy in cases of chronic arthritis, toxic myocarditis, toxic neuritis, toxic hepatic cirrhosis and toxic mental derangement, and one can recall to mind relief in the same way in many similar cases.

The above symptoms refer to the normally placed gall bladder. In rare cases there is complete transposition of the abdominal and thoracic viscera and several cases are on record of operations for acute cholecystitis on the left side.

The gall bladder sometimes extends as far as the pelvis and simulates pelvic trouble. Bland Sutton[47] reports a case in which the gall bladder was found in a right hernial sac, another in which he operated for a subserous fibroid of the uterus, but found a cancerous gall bladder attached to the fundus of the uterus and still another in which Lawson Tait operated for an ovarian cyst and found a huge mucocele of the gall bladder.

Gall bladder trouble is sometimes overshadowed by symptoms of pancreatic infection, the result of the pathological state of the former, and this can be easily understood from the experiments of Harer, Hargis and von Meter (*vide ante*),[21] which showed the very intimate association between the lymphatics of the gall bladder and pancreas.

At the beginning of the discussion of symptoms mention was made that gastric upset was a very prominent feature of gall bladder trouble; Duval, Roux. Béclère[48] from a series of radiographic studies were able to demonstrate that if periduodenitis exists before cholecystectomy, it may persist after operation; that the existence of gall stones with severe gastric disturbance suggests an accompanying periduodenitis which in some cases may be so severe as to cause duodenal stenosis from adhesions and suggest malignant obstruction.

Diagnosis.

It has to be remembered that the formation of calculi and attempts to expel them from the gall bladder and ducts are terminal events in the history of cholecystitis. Prevention is possible apparently, for McCarrison,[49] after nine years' service in the Himalayas, during which time he averaged four hundred major operations every year, did not see one case of appendicitis, one ulcer of the duodenum, one case of dyspepsia, colitis, cholecystitis or cancer.

There are many means at our command to help solve the riddle, but nothing is more helpful than an accurately and carefully compiled clinical history. Added to this, radiographic evidence is of great value, both direct and indirect: direct in that in a very small percentage of cases shadows may be got of stones in the gall bladder; indirect by pressure effects, for example, on the duodenum and stomach or by traction on and thus displacement of the duodenum, or by fixation and thus displacement of the colon.

George[50] lays particular stress on the pressure and traction effects on neighbouring viscera exerted by a diseased gall bladder in many cases. Before the advent of the dye test Edwards[51] carried out for me many of these tests and the results proved helpful. In all cases an opaque meal should be given as well as the dye and efforts made to elicit tenderness on palpation of the gall bladder when distended with dye.

In this way one may be able more easily to exclude duodenal troubles and elicit those symptoms complained of and suggestive of cholecystitis and a diagnosis in a doubtful case may be clinched.

The latest method in vogue is to give by the mouth or intravenously a solution of tetrabromphenolphthalein.

Graham[52] and his coworkers in 446 consecutive cases were able by operation to check their work, and proved the diagnosis by their dye test as correct in nearly 98%, and my results give approximately the same percentage.

Still another means devised for diagnosis and treatment is the Meltzer-Lyon test which consists of instilling a strong solution of magnesium sulphate into the duodenum by means of a tube, hoping by such means to paralyse the sphincter of Oddi reflexly to cause gall bladder contraction and thus allow collection of bile from the common bile duct, gall bladder and liver bile, and a thorough examination of each specimen, both cultural and microscopical. C. H. Mayo[53] often does this to make sure of biliary flow in jaundiced patients.

Cutler and Newton[54] carried out experiments with this test and came to the conclusion that the results for diagnostic purposes could not be depended upon, even when accurately performed.

Silverman and Menville,[55] by using this test in conjunction with the injection of tetrabromphenolphthalein, found that injections of magnesium sulphate solution produced decided reduction in the size of the gall bladder shadow and that the gall bladder was more completely drained when the duodenal mucosa was repeatedly stimulated with this solution.

The Van den Bergh test which is analogous with the tests for blood sugar and blood urea, is used to distinguish between obstructive and non-obstructive jaundice, and in this connexion Maude and S. O. Cowen[56] proved it of value in distinguishing jaundice of the obstructive type from jaundice of hæmolytic origin. We use this test extensively.

Jaundice may be present, but as a rule occurs only when there is an infective cholangitis, indirect obstruction of the common duct by a stone, or stones in the cystic duct of an acutely inflamed gall bladder or by obstruction of the common duct itself by a calculus or calculi. Too great stress is laid on the presence or otherwise of jaundice as a diagnostic sign of gall bladder infections or calculus formation; a history of its presence is helpful, but its absence by no means rules out stones or infection.

Much work has been done on the jaundice problem and hepatic efficiency.

Gerrard,[57] by using the Van den Bergh test, was able to detect latent jaundice in syphilitic

patients under treatment by arsenobenzol and found it a danger signal. Gonsalez and Karr[58] were able to achieve valuable information as to hepatic efficiency by some experiments with the so-called "hæmoclastic test"—a test which depends on the fact that in the normal individual the colloidal balance of the blood is disturbed from the absorption of proteose compounds increasing blood pressure, producing leucocytosis and other changes, whereas, when the liver cells are disturbed, the reverse effects are produced.

It is hoped that further experimentation on these lines—and there is ample room for it—will disclose functional tests of the liver similar to those carried out for estimating kidney function, more particularly in the presence of jaundice, and help considerably in deciding whether and what operative measures should be undertaken in the many gall bladder problems we are called upon to face.

Age gives some help in that stones do not as a rule occur before middle life, but in rare cases are met at a very tender age. Kennedy[59] reported the occurrence of stones in the gall bladder of a child eleven years of age. To meet them in people under thirty years of age is quite common, but often they do not make their presence felt till a ripe old age has been reached.

The symptoms themselves are the main factors helping one to a successful conclusion. Fullness, weight and distension in the epigastrium one-half to three-quarters of an hour after food, relieved by belching or vomiting, are very suggestive. The presence of flatulence not relieved by diet in a suspicious case is a very strong indication of gall bladder disease.

All the symptoms of uneasiness which force one to relax one's belt or one's clothes, brought on by certain foods, especially those of a greasy nature, with acid regurgitation, heart burn, goose flesh, are invariably present in gall bladder trouble.

Once acute inflammation sets in, Murphy's sign is almost pathognomonic and at the same time an acute stabbing pain on deep inspiration is complained of at the same spot (Boas's sign). Vomiting in these cases usually brings on severe prostration and when once an attack of colic occurs, it may be assumed that trouble has been brewing for years; and, if the colic is due to the presence of calculi, that a condition exists beyond all medical aid.

It has to be remembered that appendicitis is a very common accompaniment of cholecystitis, that ulcer of the stomach and duodenum is often an added complication, while pancreatitis is frequently present, so that the picture may be greatly obscured and add considerable difficulty in coming to a correct diagnosis of the primary seat of trouble.

Investigation of the stomach contents alone as a means to diagnosis in these cases by several experimenters has not been helpful, as hyperacidity, normal acidity and hypoacidity may be met, though we[40] have found in experiments carried out in the Pathological Department of Sydney Hospital, which we hope to publish shortly, that there is a distinct tendency towards the last condition.

The conditions most difficult to differentiate from gall bladder disease are appendicitis, gastric and duodenal perforation, renal colic, acute pancreatitis, intestinal obstruction (acute ileus), and last, but by no means the least difficult, central pneumonia and diaphragmatic pleurisy on the right side. Both of these latter conditions often accompany acute gall bladder infections and are probably due to the free lymphatic drainage of the gall bladder and to the intimate association of the sub- and super-diaphragmatic lymphatics. Anatomists do not acknowledge this anastomosis, but clinically it does exist and four and a half years ago, in support of this contention, Inglis demonstrated at Sydney Hospital a case of malignant gall bladder with myriads of secondary deposits on both sides of the diaphragm continuous with each other and passing from the peritoneal to the thoracic cavity and into the right lung.

Treatment.

Treatment should aim at preventing gall bladder infection by attending to the predisposing factors, such as constipation, bacillary infections of the urinary tract and the prescribing of a suitable diet (*a*) to avoid as far as possible any digestive troubles, (*b*) to keep the cholesterol content of the blood normal, and (*c*) prevent infection of the biliary tract by intestinal intoxication.

When symptoms of cholecystitis have arrived, administration of urotropine judicially with dieting may ward off future attacks, but by the time that stones have developed, medical treatment can achieve nothing and no more disastrous termination confronts the surgeon than cancerous formation on top of cholecystitis in which previously operation had been pleaded for.

Operations on the gall bladder are fraught with great difficulty and the bad results attributed particularly to cholecystectomy can be traced to faulty technique. The distress and pain which can be relieved by morphine only at the height of the attack, suffered in biliary colic, are considerable and one attack will make a patient weigh seriously the advantages of an operation rather than face another experience of colic. Attacks tend to follow each other closely, come on unexpectedly, each one doing considerable damage locally and to the body as a whole and, though the risks of operative procedures are not inconsiderable, operation at least offers more mercy than medicinal measures.

My endeavour has been to show you that when a gall bladder has become the seat of disease, Nature has little at her command to rid the organ successfully of its infection. As to operations on the gall bladder in general, one can say definitely that drainage of the gall bladder is becoming increasingly infrequent and removal more frequent. What operative measures will be adopted will necessarily depend on the type of case that confronts us, and

to go fully into the question would be impossible in the time at my disposal.

In jaundiced patients, and it is interesting to note that in Sherren's[60] cases of common duct stone without jaundice there was a mortality of 2·5% and with jaundice a 27% mortality, the less done operatively the better.

Before operation saline infusions and calcium chloride and even blood transfusion should be administered and checks made by the quantitative Van den Bergh test as to the amount of jaundice present. At operation local anæsthesia should be used and means adopted to keep up the bodily heat, and particularly the liver area, by warm packs, and drainage of the gall bladder or common duct instituted, preferably the former. After operation fluids must be given freely and the bodily heat maintained and, if necessary, blood transfusion again used.

Inhalation anæsthesia must be studiously avoided, as jaundiced patients bear ether anæsthesia in particular badly.

If malignant disease is obstructing the common duct and cholecyst-enterostomy is contemplated, preliminary drainage should be carried out. As a rule these cases come too late and suffer a high mortality rate. It is of interest that Walters[61] advocates preliminary drainage.

In non-jaundiced patients, in the absence of complications, cholecystectomy without drainage is the operation of choice, even in the presence of pancreatitis, for it is my firm conviction, after operating upon many hundreds of these patients, that once a gall bladder becomes infected, it will remain infected and be a source of danger to the patient till removed.

No patient has yet come under my observation whose gall bladder has been removed correctly, the presence of which would save or be of value in saving his life or improving his health. On the other hand, many patients have consulted me with trouble from a gall bladder previously drained, with complete restoration to health after its removal.

Let us take analogy from the urinary bladder. Cystitis will clear up after removal of a calculus, where there is no obstruction to the outflow of urine, such as by stricture or enlarged prostate, but in an infective condition, even with constant medication internally and externally, practically never. What hope is there, then, for the gall bladder with its walls eaten into, disabled by adhesions to adjacent structures, with a tortuous and perhaps a sclerosed and narrowed duct, of ridding itself of infection?

In suport of this are the results of the investigations of Jordan[62] with typhoid fever in the Rhine Province, where it was observed that a large proportion of all persons suffering from gall stones were discharging typhoid bacilli in their stools. These disappeared completely from the stools after cholecystectomy, except in a few patients with infected spleens and bone marrow to keep up the supply.

The typhoid bacillus is by no means a resistant organism and thus the tenacity of an ordinary pathogenic infection of the gall bladder can be appreciated, more particularly when from a gall bladder removed by me a very active culture of typhoid bacilli was grown fifteen years after the primary infection. (At Saint Vincent's Public Hospital, 1920, not reported.)

In the majority of cases of calculus cholecystitis, ulceration has been going on for years, more or less stenosis of the cystic duct gradually has taken place with considerable injury to the gall bladder itself, and by using drainage only one is laying up trouble for the patient and incidentally perhaps for oneself.

Many surgeons of high repute still recommend drainage as a routine, but those most experienced and skilled in this region adopt the radical operation and use the minor performance for the more debilitated individuals or as a temporary measure in cases of jaundice.

Bland Sutton[63] boldly says: "To operate for acute cholecystitis and not remove the gall bladder is as reprehensible as operating for gangrenous appendicitis and leaving the appendix." After such a celebrated authority one may be pardoned for hesitating to express an opinion, but my feelings are that a diseased gall bladder is never an asset, but a dangerous liability and never more so than when acutely inflamed.

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Reports of Cases.

TETANUS.

By Idris Morgan, M.B., Ch.M. (Sydney),
Honorary Physician, Newcastle Hospital, New South Wales.

A boy, aged ten and a half years, was admitted to Newcastle Hospital on June 8, 1930. He became ill five days prior to admission. On June 7, 1930, he complained of stiffness of the jaws and a diagnosis of tetanus was made.

The boy stated that a nail had punctured the sole of his foot "about three weeks" before he became ill. A healed punctured wound was found on the sole of the right heel.

On admission a typical and well marked *risus sardonicus* was present. He complained of pain in the back, neck and right groin. The jaws were tightly clenched. He was able to speak and was quite rational. Frequent spasms occurred, during which the body became arched to such an extent that it rested in the head and heels. The muscles of the neck, back and jaws were very rigid, the arm muscles were rigid, but voluntary movements could be made. The right leg muscles were more rigid than the left, but both legs could be moved. At this stage the abdominal muscles were not rigid. Speech and deglutition were never affected throughout the illness. The *rectus abdominalis* muscles became rigid later and, as is often the case, still show some rigidity (June 23, 1930), when all other muscles have returned to normal.

The temperature on admission was 40° C. (104° F.) and the pulse 145. The illness was not accompanied by much fever, except on the ninth and tenth days (38·9° C. or 102° F.), when an acute erythematous serum rash made its appearance. The patient made a complete recovery.

Serum was given for nine days. Morphine was used freely to overcome the muscular spasms and to combat the

extreme restlessness. Eggs and milk were fed between the clenched teeth and were swallowed well. The usual precautions (darkened room, dark bed cover *et cetera*) were taken to exclude stimuli which might have commenced a spasm. The accompanying table shows the dosage of the serum employed, route of injection and comments on progress.

Date.	Dosage and Route, Units.	Comments.
June 8, 1930. Admitted 2 p.m.	20,000 Intravenously 15,000 Intrathecally 5,000 Around site of wound	Opisthotonos position assumed during manipulations incident to injection. Morphine given.
June 9, 1930.	20,000 Intravenous 10,000 Intrathecal	Under influence of morphine. Fewer spasms. Rigidity of muscles unchanged.
June 10, 1930. a.m. ..	20,000 Intravenous 10,000 Intrathecal	General condition moderately good. Morphine continued to control restlessness and spasm.
p.m. ..	20,000 Intravenous	
June 11, 1930. a.m. ..	20,000 Intravenous 20,000 Intrathecal	Rigidity of muscles still marked. Fewer spams. Restless.
p.m. ..	20,000 Intravenous 20,000 Intrathecal	
June 12, 1930.	As for previous day	Improved. Slight voluntary anterior flexion of vertebral column observed. Still very rigid. Arms held in position of flexion and stiff.
June 13, 1930	As for previous day	General condition improved. Slight separation of teeth possible.
June 14, 1930	As for previous day	Marked improvement. Jaws separated voluntarily ¼ inch. Serum rash. Temperature 38·9° C. (102° F.).
June 15, 1930	As for previous day	Further improvement. Mouth opened 1 inch. Serum rash marked. Temperature 38·3°C. (101° F.).
June 16, 1930	20,000 Intrathecally	Mouth open well. Rigidity of all muscles almost disappeared. Serum ceased. Serum rash fading. Temperature 38·3° C. (101° F.).

On June 15, 1930, the cerebro-spinal fluid was turbid. Microscopical examination revealed "pus cells" but no organisms. No signs of meningeal irritation were observed. The serum was given on June 16, 1930, intravenously to replace the cerebro-spinal fluid withdrawn for examination. The temperature fell on June 18, 1930, and has been normal since.

Comment.

It is well known that the tetanus bacillus sends a virulent toxin into the blood stream from the site it has occupied in the body. It is also generally assumed that the serum used to combat this disease has no bactericidal action, but merely neutralizes the toxin and so prevents death till the defensive mechanism of the body has overcome the invading organisms. It is logical to assume that while symptoms attributable to the toxin continue to be manifest, the organisms at the site of the invasion have not been destroyed and the amount of antitoxin injected has not been sufficient to neutralize the toxins given off by them. It therefore follows that the dosage should be increased, regardless of the actual number of units given, till there is definite clinical evidence that the toxins are being neutralized and that dosage continued till the physician is sure, from the clinical signs, that no more toxins are being sent into the blood stream. Text books recommend doses which to me appear to be woefully inadequate. The dosage of antitoxic serum, it seems to me, should be increased or decreased, according to the results which follow its injection.

It would appear that diphtheria antitoxin acts in the same way as that prepared for use in tetanus. If this be so, why does the membrane shrivel so quickly? The serum, it is said, has no antibactericidal power. Is the membrane a manifestation of a reaction to toxins and not to bacteria, and does the initial dose of antitoxin neutralize all the toxin present in the blood stream and tissues and provide sufficient surplus to deal with any further toxins that may be given off, while the organisms still remain and multiply in the fauces, as they do for weeks in some cases?

In tetanus it appears that antitoxin must be given day after day in large doses to neutralize a continuous stream of toxins given off from the site of infection till the tetanus bacilli are destroyed, presumably by phagocytic action. (Why is there so often no evidence, swelling, suppuration *et cetera*, of this local defence mechanism?)

In diphtheria a single injection of antitoxic serum apparently overcomes the whole infection, yet the sera are supposed to act in the same way. I have been unable to find any enlightenment in the literature on this point. Hewlett, writing in the Medical Research Council's "System of Bacteriology," postulates a theory that natural immunity to diphtheria is acquired by repeated infections with avirulent strains of diphtheria bacilli which increase the antitoxic content of the blood.

Persons manifesting clinical signs of diphtheria obviously have little if any antitoxin in the blood at the time of infection, and Glenny and Sudmessen found that if diphtheria toxin were injected into an animal not previously treated, a latent period of three weeks would elapse before antitoxin could be detected and the maximum antitoxin content would not be reached until about two months after the injection. Again, then, why does the single dose of antitoxic serum shrivel up the membrane and render the virulent organisms still present in the throat harmless within twenty-four hours in the case of diphtheria, while in tetanus and other diseases in which antitoxic serum is used, the same result is not obtained unless the serum is continued in large, repeated doses as long as there are signs that indicate that the invading organisms are still alive at the site of invasion and sending toxins into the blood stream.

Apropos of this discussion, it would seem that antistreptococcal and antipneumococcal serum have been given a worse reputation than they actually deserve, for this very reason, that larger doses are not used at frequent intervals and continued while clinical signs indicate that the invading organisms have not been overcome by the defensive mechanism of the body. There seems to be little fear from the amount of serum injected.

TETANUS, ITS SPECIFIC AND GENERAL TREATMENT: A SUMMARY OF SEVENTEEN CASES.

By Harley Stevens, M.B., Ch.M. (Sydney),
Resident Medical Officer, Children's Hospital, Perth.

The interesting report of Dr. Paterson from the Hospital of Sick Children, Brisbane, on the results of treatment of tetanus with specific antitoxins has inspired me to compare his figures with a series receiving rather smaller doses of the same serum.

For this purpose the histories of all the patients with tetanus treated at the Children's Hospital, Perth, within the last five and a half years, have been summarized in the accompanying table in a manner similar to Dr. Paterson's series.

Unfortunately the seventeen cases form a very small group from which to draw conclusions. Nevertheless a surprising similarity in the two series is evident.

Of the five patients who died, giving a mortality of 29·4%, three died within twelve hours of admission. The fourth (Case X) was complicated by an old rheumatic history and

TABLE I.

Showing a Summary of Seventeen Consecutive Cases of Tetanus.

Case Number.	Age in Years.	Incubation Period.	Time since Onset.	Units of Antitoxin.					Remarks.
				Intravenous.	Intramuscular.	Intrathecal.	Subcutaneous.	Total.	
1	5	7 days	1 day	4,500	24,000	5,000		33,500	Cured.
2	10	10 days	1 day	3,000	20,000	",000	102,000	132,000	Cured.
3	11	16 days	5 days	10,000	30,000	7,000	3,000	50,000	Cured.
4	12	6 days	1 day		20,000	8,000		28,000	Cured.
5	6½	7 days	2 days	13,000	52,000	7,500		72,500	Cured.
6	4	18 days	3 days	23,000	5,100	2,000		76,000	Cured.
7	6½	?	1 day			3,000	6,000	9,000	Died within twelve hours after admission.
8	4	2 days	1 day		15,000	5,000	20,000	40,000	Died within twelve hours after admission.
9	8	10 days	2 days	18,000	60,000	10,000		88,000	Cured.
10	10	5 days	1 day		55,000	30,000		85,000	Died four days after admission. Cardiac failure. Old history of chorea.
11	5	28 days	?		20,000	20,000		40,000	Died within twelve hours of admission.
12	5	4 days	3 days		14,500	11,500	108,500	120,000	Cured.
13	11	7 days	1 day		2,000	2,000		4,000	Died three days later. Diabetes.
14	8	7 days	3 days		40,000	14,000	2,000	56,000	Cured.
15	8	?	3 days		64,000	40,000		104,000	Cured.
16	11	?	3 days	15,000	45,000	20,000		80,000	Cured.
17	10	5 days	2 days		110,000	40,000		150,000	Cured.

TABLE II.

Showing Days on which Injections were given to Seventeen Consecutive Cases of Tetanus.

Case.	1st Day.	2nd Day.	3rd Day.	4th Day.	5th Day.	6th Day.	7th Day.	8th Day.	9th Day.	10th Day.
1	+	+	+							
2	+	+	+	+		+	+			
3	+			+		+				
4	+									
5	+		+	+						+
6	++	+	+		+					
7	+									
8	+									
9	+	+				+				
10	+	+	+	+						
11	+									
12	+	+	+	+	+	+				
13	+									
14	+	+	+	+	+					
15	+			+						+
16	+							+		
17	+	+								

died after four days with signs of cardiac failure. In the fifth (Case XV) there is a considerable probability that the condition was one of diabetes with convulsions and coma. An initial dose of 4,000 units was given and the treatment not persisted with.

The urine contained sugar and acetone and the blood sugar was considerably raised; but the cause of death is given as tetanus.

Diagnosis.

There is nothing to add to Dr. Paterson's remarks in this regard save further to stress the early appearance and frequency of board-like rigidity of the abdominal wall with only moderate tenderness and a certain amount of movement with deep respiration.

General Treatment.

Dr. Paterson has enumerated the usual routine points of treatment. Of sedative drugs, the bromides and chloral hydrate were given at regular intervals in each case. Chloretone appears to have been the next favourite, while morphine in doses up to one-twelfth of a grain was given hypodermically to eight patients, three of whom survived.

Calcium lactate was tried in a few cases by mouth, but there is no mention of magnesium sulphate intrathecally.

Specific Treatment.

No routine method of administration of antitoxin is disclosed in the histories, although in every case the initial dose was given into the spinal theca.

In three instances (Cases III, X and XVII) the intrathecal injection was repeated and in Cases X and XVII marked turbidity of the cerebro-spinal fluid was noted at the second puncture. In Case XVII this was due to great numbers of leucocytes in which the mononuclear type predominated, being possibly a reaction to the first injection.

The largest amount given into the theca in one injection (Case XV) was 40,000 units. One would hesitate before making so large a dose in that site a routine procedure, but the patient, who was quite a severe case, with marked rigidity and many spasms, did remarkably well.

The bulk of the serum has been given into the muscles. Intravenous and subcutaneous injections were often omitted, though in a large proportion of children it should be a simple matter to inject antitoxin in the same manner as a dose of "Novarsenobillon" is given.

The total cost of antitoxin in any single case was less than £17. The Commonwealth Serum Laboratories' product was used in all cases and the units were the American rating.

Frequency of Injection.

In only one case (Case VI) were injections given more than once in the day. In his case intravenously on the first day of treatment.

Injections rarely extended beyond the sixth day of treatment, but Patient II received a dose of antitoxin on the seventh day, Patient XVI one on the eighth day, and Patients V and X one each on the tenth day.

Chloroform anæsthesia, preceded by a full hypodermic dose of atropine, has been employed as a routine for lumbar puncture and injection of antitoxin.

There were fifteen boys and two girls in the series.

One patient admitted as suffering from tetanus and discharged with the same diagnosis, is not included because of lack of definite evidence of the nature of the complaint. The history of cutting his foot while digging in the garden two weeks before admission probably influenced the staff so that it was thought advisable not to temporize, and 40,000 units were given into the muscles. The boy

aged ten years, complained of pain and twitching in the affected leg. There was an infected wound of the foot and some inguinal adenitis. The mother's statement that the arms and neck were also "twitching" was not confirmed after admission. There was no trismus.

One does not attempt conclusions on the results, but they do leave some doubt in one's mind of the necessity for the administration of a million or more units of antitetanic serum to any one patient with tetanus under the age of thirteen years.

The cost of antitoxin is a very real problem in many hospitals, but further evidence with regard to massive dosage will be eagerly awaited.

It is unearthing an old bone of contention which was well worried during the war, without any definite conclusions being reached with regard to the optimum quantity or the site for injection.

From the less laboured aspect of civil practice in children, however, one may be pardoned for presenting the foregoing figures.

Acknowledgement.

I am indebted to Dr. W. Seed, Medical Superintendent, for permission to publish these cases.

TUBERCULOSIS OF THE KNEE: TREATMENT WITH TUBERCULIN.[1]

By Guy Griffiths, M.D. (Sydney),
Senior Honorary Physician, Royal North Shore Hospital of Sydney.

E.W., a woman, aged twenty-five, was admitted to the Royal North Shore Hospital of Sydney under my care on July 20, 1929, complaining of swelling and pain in the left knee for three years and of cough and loss of weight for seven months.

Her father had died of heart failure; her mother, brothers and sisters were all alive and well. She was a book-binder, but had not been able to work for three months.

Her present illness began with pain and swelling of the left knee three years ago; she now attributes this to a fall she remembers she had at that time. The pain and swelling have become worse during the last four or five months. For the last seven months she has coughed, especially at night, expectorating sputum which has sometimes contained small amounts of blood. She has lost weight considerably, although she has a moderately good appetite. The patient lay comfortably in bed in the dorsal decubitus. Her cheeks were flushed. Her tongue was clean, her teeth good, her fauces clear. In the abdomen the pregnant uterus was palpable just above the umbilicus. There were no morbid abdominal signs. The pulse was regular, 86 to 96 per minute. The temperature was normal and the respiration rate 20 per minute. The apex beat was in the fifth space 8·75 centimetres (three and a half inches) from the middle line. The cardiac dulness extended from the third rib above and the edge of the sternum on the right to the apex beat. The sounds were clear. The chest moved evenly on respiration, the expansion was moderate, the percussion note slightly impaired at the right apex in front and at the back and possibly also at the left apex behind. The breath sounds were feeble and slightly high in pitch at the right apex. The vocal resonance was not increased, no râles were heard.

The urine was acid, of specific gravity 1016, and contained neither albumin nor sugar. The left knee was greatly swollen, but not red. The swelling extended above and below the joint; a little fluid was present. The patient could not bear to bend the knee, but held it extended. A skiagram of the knee showed some erosion of the tibial spine and diminution of the joint space and blurring around the joint. The honorary radiographer considered this due to infective arthritis, probably tuberculous. With this opinion Dr. Coppleson, who kindly saw her in consultation, concurred; he recommended that the joint be kept in plaster of Paris, fully extended, in an attempt to induce ankylosis.

This was done, but as her pregnancy progressed, great swelling appeared in the left foot, ankle, leg and thigh, as well as around the knee, and the pain increased so that we were compelled to remove the plaster and to allow her to flex the knee slightly, as full extension now increased the pain.

Albuminuria occurred, but not to an alarming extent.

Numerous patterns of splints were tried, but she could bear none of them, and the œdema of the left leg increased.

Meanwhile she was continuously in bed on the ward veranda, taking light diet with extra eggs and milk, an alkaline gentian mixture with 0·6 mil (ten minims) of tincture of *nux vomica* three times a day before meals and increasing doses of albumose-free tuberculin, beginning on July 31 with 0·00001 cubic centimetre and increasing twice a week up to 0·0014 cubic centimetre on October 1. She had a slight local reaction in the arm at the site of the injection to most of these doses, but no general and no focal reaction. The cough and sputum diminished, no tubercle bacilli were found in any of seven microscopical examinations, but the increasing abdominal tumour and the swelling and pain of the left knee caused considerable distress.

She was transferred to the Obstetric Department for her confinement, which happily was conducted without particular difficulty or complication.

On her return to the medical ward at the beginning of November, 1929, I resumed treatment as before, including the tuberculin, and have since been able to increase the dose until on June 1, 1930, she had 0·68 cubic centimetre of old tuberculin (of Burroughs Wellcome manufacture) without any sign of reaction. To some of the intermediate doses she had reacted, often with soreness in the arm at the site of the injection, occasionally with slight fever, for example, to 37·5° C. (99·6° F.), headache and malaise. As general œdema of the leg did not recur, we were able to put her knee in a long plaster for four months. Early in June this was replaced by a lighter one and she now walks moderately well with two crutches. She has no cough and no sputum, but there is a little dulness with feeble broncho-vesicular breathing at the right apex. She is stout and ruddy, and her weight has increased to 79·2 kilograms (twelve stone eight pounds).[1]

[1] The patient herein described was shown at a meeting of the New South Wales Branch of the British Medical Association on June 12, 1930.

[1] A hypodermic injection of 0·75 cubic centimetre of old tuberculin was given during the meeting.

Reviews.

HYGIENE.

Dr. J. R. Currie, Professor of Public Health in the University of Glasgow, has attempted to provide "A Textbook of Hygiene" for the medical student taking the ordinary curriculum for a degree in medicine as well as for the graduate aspiring to a diploma in public health.[2]

In the latter regard, however suitable the book is for the qualifications in Great Britain, sanitary law being quoted for both Scotland and England, it cannot be commended to students in Australia.

The legal material being included in each section along with the subject to which it relates, whilst possibly an advantage in teaching English and Scottish students, will certainly be confusing to Australians who have to deal with the public health legislation of the Commonwealth and the States.

Recognizing that the principles underlying preventive medicine must be made the inspirational leaven of the whole training of the future medical practitioner, one

[2] "A Textbook of Hygiene," by J. R. Currie, M.A., M.D., D.P.H., M.R.C.P.; 1930. Edinburgh: E. and S. Livingstone. Demy 8vo., pp. 864, with 110 illustrations. Price: 27s. net.

would suggest that in future editions the book might be issued in two sections, one dealing with personal hygiene, infectious diseases, maternity and child welfare, school hygiene, industrial hygiene and food; and the other with public health administration, sanitary law and those subjects more especially required for the diploma of public health.

The chapters on air and ventilation, warming and lighting, soil, water supply, refuse and sewage disposal and rivers pollution are well written and can be commended as being in accord with modern research and experience.

Under "Miscellanea" are included national health insurance, medical certification, aids to medical practice, and health propaganda, which are now features of public health administration.

The book is one worthy of a place in any health department reference library, but from the point of view of the Australian student is liable to make the present confusion of enactments even worse confounded.

THE RADIOLOGICAL DIAGNOSIS OF PEPTIC ULCER.

"Peptic Ulcer," by Jacob Buckstein, the tenth volume of the *Annals of Roentgenology*, falls rather short of the high standard of excellence of previous volumes of this series of monographs.[1] The author, a man of wide experience, must have been confronted on innumerable occasions with cases that presented difficulty in diagnosis—very early lesions, atypical lesions and cases with complications. These are hardly touched upon and practically the whole book is devoted to cases which are so typical or so advanced that only a novice could experience difficulty in the diagnosis.

The book commences with a brief general description of peptic ulcer and then passes on to a more detailed consideration of gastric, duodenal and gastro-jejunal ulcers, illustrated by numerous examples. These cases are well presented, a brief clinical history being followed by surgical and *post mortem* results and pathological and X ray findings. Unfortunately many of the skiagrams are of poor quality. The chapter on gastro-jejunal ulceration is very good indeed and a few excellent cases are presented.

The greatest difficulties that confront the radiologist in the diagnosis of gastric ulcer, lie in the differentiation of true ulceration from other conditions which deform the contour of the stomach, such as carcinoma, spasm, adhesions *et cetera*. The author, dealing only with peptic ulcer, does not fully discuss these other conditions and herein lies an inevitable weakness.

However, although the book covers only a limited field, this field is a large one and the book can be recommended to anyone who is desirous of acquiring a general knowledge of the radiological diagnosis of peptic ulcer.

RADIOLOGY OF THE URINARY TRACT.

"Urological Roentgenology" forms the seventh volume of the *Annals of Roentgenology*, as edited by Dr. James T. Case.[2] The authors point out that the great progress made during the last decade in the diagnosis of surgical conditions of the urinary tract is in large part due to the improvement of methods of X ray examination. There is now in fact no subject in diagnosis in which X rays play a more important part than in the field of urology, and there is no field in which greater skill is required for the exact interpretation of X ray findings. The competent urologist will have at least as great an opportunity to master this subject as the Röntgenologist, and this book is an attempt to correlate the work of each.

The value of the volume is very greatly enhanced by the *précis* of the clinical histories which is appended to many of the urograms.

While perhaps the opening chapters dealing with various types of equipment and topography, anatomy and physiology of the urinary tract could have been curtailed or even omitted with advantage, there is little room left for adverse criticism in the manner or method of presentation of the remainder.

We could, however, have wished that a little more attention had been paid to pyeloscopy. Those who have employed it will certainly not agree with the authors' statement that pyeloscopy "has little to add and very distinct disadvantages." It is certainly essential for the accurate diagnosis of the neuro-muscular disturbances of the pelvis, calyces and ureter which are becoming of increasingly recognized importance.

The volume, on account of its price, will not find a very wide circle of readers, but will be useful to those who require a work of reference of this type.

[1] "Annals of Roentgenology: A Series of Monographic Atlases," edited by James T. Case, M.D.; Volume X, "Peptic Ulcer," by Jacob Buckstein, M.D.; 1930. New York: Paul B. Hoeber, Inc. Demy 4to., pp. 360, with two hundred and forty Roentgen ray studies and forty-seven clinical illustrations. Price: $12·00.

[2] "Annals of Roentgenology: A Series of Monographic Atlases," edited by James T. Case, M.D.; Volume VII: "Urological Roentgenology," by Hugh H. Young, M.D., and Charles A. Waters, M.D.; 1928. New York: Paul B. Hoeber, Incorporated. Demy 4to., pp. 544. Price: $20.00 net.

Analytical Department.

"HEPABOS" AND "PORCULIN."

"Hepabos" and "Porculin" are the names of two products of a firm known as Glandular Preparations, Limited; they are prepared respectively from ox livers and hog stomachs and it is claimed that they contain the active principle or principles efficacious in the treatment of Addisonian or pernicious anæmia.

An inspection has been made of the premises of the firm and the process of manufacture has been investigated. The cleanliness of the operations was of a high order. The livers and hog stomachs are passed by government authorities and the solvent is of a quality commonly used commercially. The frozen livers are allowed to thaw, any adherent fat or blood vessels are removed, they are weighed, minced and added to acetone in a bin. The minced liver is agitated in the acetone. The acetone is then run off and the liver substance is put into shallow pans and dried *in vacuo*. It is then sieved to remove fibre and the resulting powder is the product intended for the market. It may or may not be flavoured. In either form the product has very little taste or odour.

In order to determine whether any active principle had been lost by solution in the acetone residues the following experiment was undertaken. The acetone residue from sixty pounds of liver *plus* sixty pounds of acetone, which amounted to ninety-three pounds, was evaporated down *in vacuo* at a temperature not exceeding 100° F.. The residual liquor was then 1,400 cubic centimetres and, of course, would contain the whole of the active principle which was dissolved out with the acetone. To this residual liquor was added alcohol so that the percentage of alcohol was 70, in which percentage the active principle is readily soluble. The mixture was agitated each day for four days and then filtered. The precipitate was rejected and the filtrate was divided into three lots. One lot was made up to 95% alcohol and was violently agitated at hourly intervals for six hours. A second lot was made up to 95% alcohol, kept at 37° C. and was violently agitated for six hours. The third lot was made up to 96% alcohol and kept at room temperature and violently agitated for some hours. In none of these three portions had any precipitate developed after some days. It can thus be claimed that the acetone dissolves no substance which is insoluble in 95% alcohol, but soluble in 70% alcohol.

According to clinical reports satisfactory results have been obtained in pernicious anæmia by the use of "Hepabos" and "Porculin." Sometimes these have been used in a mixture of 75% of the former and 25% of the latter. These substances may be regarded as reliable products and suitable for use in the treatment of Addisonian anæmia. "Hepabos" is also marketed in tablet form under the trade name of "Sang Cre."

The Medical Journal of Australia

SATURDAY, OCTOBER 4, 1930.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction, are invited to seek the advice of the Editor.

MEDICAL PRACTITIONERS AND LITIGATION.

A medical practitioner called upon to attend an individual suffering from an illness or from the effects of an injury accidentally received, is expected first of all to be possessed of knowledge that will enable him to discover the nature of the illness or the extent of the injury and, secondly, to display skill in the institution of treatment appropriate to the condition. Should the diagnosis be obscure (and medical practitioners know only too well that diagnosis is sometimes, though rarely, a matter of such difficulty as to be well nigh impossible), it is to be expected that symptoms will be countered as they arise and that the patient will be so placed and treated that the natural resistance of his body tissues will be at its best and that the advent of distinctive signs or symptoms will not pass unnoticed. It is obvious that, since medicine is not an exact science, difference of opinion in regard to a diagnosis will sometimes exist and not infrequently the treatment advocated by one medical practitioner will appear to be divergent from that suggested by another. In these circumstances both practitioners would interpret the symptoms in the light of their experience and would act accordingly—both would be exercising reasonable care. Since reasonable skill and care are expected of a medical practitioner, it naturally follows that lack of skill, want of care and negligence would be adequate grounds for complaint by a patient and the patient would be justified in claiming damages in a court of law.

During recent months several legal processes have been set in motion against medical practitioners in different parts of the Commonwealth. The grounds for these actions have been of a varied nature and many matters of practical importance have been raised. It must be clearly understood that it is not our intention to discuss the rights or wrongs, the motives or justifications, in any of these cases. Because, however, the occurrence of so many legal actions is fraught with danger to the standard of medical practice, the duty of the medical practitioner must be made clear. The danger arises from the fact that a medical practitioner's desire to protect himself against litigation may come into conflict with his clear duty to his patient. If he allows the former to predominate, it is possible that but few will blame him, but he is undoubtedly doing wrong. For example, when a limb has been damaged to such an extent that it is doubtful whether it can be saved, desire for his own protection may lead the medical practitioner to undertake amputation as the proper treatment—he may consciously or unconsciously persuade himself that amputation is the best treatment. He may argue that if he succeeds in saving the limb and the result is not anatomically perfect, he might be called upon to defend a legal action. It will be a bad thing for surgery if fear of litigation results in an increase in the number of amputations. Already some surgeons state that in certain circumstances they refuse to treat patients with fractures. Again, if a medical practitioner for fear of legal attachment refuses to certify as insane a person who should be under institutional control, the community will suffer.

The duty of the medical practitioner is clear. He has his duty to his patient and he has his duty to himself and his profession. The patient must be treated along lines which will enable him to be most

useful in the future, regardless of possible personal consequences to the medical practitioner. The duty to the patient must be preeminent. At the same time the practitioner should protect himself as far as possible. He may do this in some instances by explaining the matter fully to the patient or his relatives, in some by consultation with his colleagues. Unfortunately, too, there are in any community litigious persons who regard a medical practitioner as a good mark, who look for circumstances that may be turned to good account, and who, having found them, start a legal action in the hope that it will lead them to affluence. A medical practitioner may best thwart these litigious persons by belonging to a defence association; money-seekers think twice about undertaking a chance action against such a body. It should be pointed out, that, though a medical practitioner succeeds in winning an action at law and though all his costs may be paid by an association existing for that purpose, he undergoes a period of acute mental stress when faced with such a proceeding. Some men find it very difficult to recover their judgement and sense of proportion after facing an unjustified legal action. Patients should be taught that a functionally perfect limb is better than an anatomically perfect one that is not so useful. In regard to lunacy certificates, the law might be amended so that recertification by medical officers of the Department of Lunacy, carried out soon after the patient's reception, would absolve the original certifying practitioners of liability. In these and other ways it should be possible to lessen the regrettably large number of legal actions.

Current Comment.

ACHALASIA OF THE CARDIA.

DILATATION of the œsophagus without organic obstruction has been recognized for longer than a century, but the cause of the condition has never been quite clearly understood. Since the work of Mikulicz and Meltzer in 1888 muscular spasm of the œsophagus has been the most widely accepted explanation of the ætiology. This is still the most popular theory despite its flaws, included among which may be mentioned the failure to supply a reason for the spasm. The term cardiospasm has been generally adopted.

There has been some controversy regarding the mechanism of deglutition and the innervation of the œsophagus. The œsophagus is supplied throughout its length by the vagus nerve whose fibres terminate in the ganglia of Auerbach's plexus. The existence of a cardiac sphincter has been denied by some, but there can be no doubt that the musculature of the lower end of the œsophagus functions similarly to sphincter muscles elsewhere. Though there has been disagreement on the point, it is probable that the sphincter receives sympathetic innervation in addition to vagal fibres. Stimulation of the vagus causes muscular contraction in the thoracic portion of the œsophagus, but relaxation of the sphincter. The sympathetic fibres are concerned in sphincteric contraction. It has been said that the sphincter remains closed by reason of chemical stimulation by the acid contents of the stomach. This scarcely seems reasonable when it is considered that the stomachs of many otherwise normal individuals contain no acid.

Acute and chronic types of cardiospasm have been described. The condition has been regarded by some as invariably due to hysteria, others again have considered that while acute spasm may have an hysterical basis, the chronic type probably has some organic nervous cause. Kinking of the œsophagus, contraction of the diaphragmatic muscular fibres surrounding the *hiatus œsophageus*, pressure and distortion by the liver, reflex spasm following œsophagitis *et cetera*, have been from time to time suggested as causes of blockage of the cardia with its accompanying œsophagectasia. None of these theories has been quite satisfactory.

Though he was preceded by Einhorn in 1888 and Rolleston in 1896, Arthur F. Hurst in 1913 drew attention to the possibility that the term "cardiospasm" was a misnomer and that the condition was due rather to a failure of the cardiac sphincter to relax, and later he suggested as more suitable the name "achalasia of the cardia," meaning absence of relaxation. Recently Hurst and Geoffrey W. Rake in an interesting paper have reviewed the subject and presented their conclusions as to ætiology, pathology and treatment.[1] They do not deny the possibility of the occurrence of true cardiospasm due to stimulation of the sympathetic nerve supply and point out that there are three other possible abnormalities of the cardiac sphincter, namely, a patulous condition due to vagal overaction, dilatation due to sympathetic destruction, and inhibition of relaxation due to vagal destruction. They think it is very doubtful whether cardiospasm ever occurs as a purely functional disorder and remark that in their experience hysterical dysphagia affects only the voluntary part of the act of deglutition. When a condition of achalasia of the cardia exists, food is retained in the œsophagus and violent peristaltic efforts are made to overcome the obstruction. These efforts are at first successful, but later dilatation develops and though the circular muscles hypertrophy greatly, continuous

[1] *The Quarterly Journal of Medicine*, July, 1930.

violent peristaltic movements are unable to exert sufficient pressure on the sphincter and do nothing more than churn the stagnating contents of the œsophagus. The sphincter opens only when forced by the weight of the food within the œsophagus, that is, when the column is greater than 20·32 centimetres in height. This may be readily observed by means of X rays and the opaque meal. There is no hypertrophy of the cardiac sphincter, a further argument against spasm.

Hurst and Rake believe that the primary ætiological factor is an inflammation of the vagal ganglia of Auerbach's plexus at the cardiac end of the œsophagus. In the later stages the ganglia are completely destroyed and replaced by cicatricial tissue. As the disease progresses the ganglia are affected higher up the œsophagus so that the muscle tone normally maintained by the vagus is lost. This loss of tone is responsible for the extreme dilatation which is far greater in this condition than when organic obstruction is present. As a result of increase in length which occasionally occurs, the œsophagus may be forced to take a curved course through the thorax. Sacculation is then likely to occur. Chronic inflammation and ulceration of the œsophagus inevitably result when the condition is of long standing. Epithelial hyperplasia, leucoplakia, wart-like nodules and carcinoma are numbered among the complications.

It is difficult to reconcile one or two of the facts concerning so-called cardiospasm or idiopathic œsophagectasia with the views of Hurst and Rake. In the first place there is the sudden onset. This may be explained perhaps by remarking that no symptoms are experienced until obstruction is made suddenly complete by the advent possibly of some local inflammation of the mucous membrane. Another apparent anomaly is the violent peristalsis despite the destruction of the ganglia of Auerbach's plexus, but it has been shown experimentally that peristalsis, though inhibited by division of the vagi, recommences after a period. It also may be asked, if achalasia of the cardia is due to an organic nerve lesion, why should it be cured by dilatation as stated by Hurst and Rake? Does dilatation result in complete paralysis of the sphincter so that it remains permanently open? It is difficult to conceive how dilatation may reeducate a sphincter whose ability to relax has been lost through destruction of its nervous mechanism. The institution of a myogenic control scarcely seems possible while the sympathetic innervation remains intact and retains its activity. Paralysis of the sympathetic nerve endings as a result of dilatation is a remote possibility. It would appear that the simple explanation presented by Hurst and Rake requires some amplification.

OBSTETRICS AT GUY'S HOSPITAL.

In obstetrics analysis of results in any one clinic is one of the methods by which some estimate may be formed of the value of a method of treatment. In a modern obstetric hospital records are, or should be, complete, but it must be remembered that the patients admitted to an obstetric hospital include many with abnormalities. The obstetric practice of such a hospital thus gives no indication of the number or proportion of abnormal conditions likely to be found in private practice. In a recent report of the work carried out in Guy's Hospital Maternity Department in 1929 a method different from that usually followed has been adopted.[1] The patients have been considered in two groups. Group A includes all patients (1,575 in number) ordinarily living in the Guy's Hospital maternity district, whether delivered in their own homes or in the hospital wards and whether they attended the antenatal clinic or not. Group B includes all patients living outside the hospital district (106 in number). G. F. Gibberd, the Obstetric Registrar, who writes the report, is probably justified in his claim that Class A represents a remarkably unselected group of women. This method of preparing the report is one that might be adopted in other places where the environment of the hospital makes it possible.

It is not possible to discuss the whole of Gibberd's report. His figures for maternal mortality and some of the points raised by him in discussion are of interest. Among patients in Class A there were four deaths, 0·26%. Two patients died of paralytic ileus after Cæsarean section. One of these operations was done for concealed accidental hæmorrhage and one for a contracted pelvis. One patient died from primary pneumococcal pneumonia in the puerperium; there was no uterine infection. The fourth death was that of a patient confined after normal labour in the district. The patient died of septicæmia as a result of a uterine infection with *Streptococcus pyogenes*. One of the attendants of the last patient was found to have *Streptococcus pyogenes* in his throat without any signs of inflammation. A series of puerperal infections of a somewhat similar origin was reported in this journal by Marshall Allan and Bryce in March, 1928. The two points emphasized by Gibberd are the prevention of the "first case" and the prevention of spread of infection when the first case is discovered. The "first case" in a public hospital would generally be due to the admission of an infected patient who was advanced in labour. Gibberd's record of the infective attendant shows that this source of infection must not be forgotten. As far as the private medical practitioner is concerned, the "last case" must be remembered—the last infective person with whom the medical practitioner has come into contact. In this connexion emphasis must be laid on the necessity for careful bacteriological investigation; both Marshall Allan and Bryce, and Gibberd have shown how important and useful it is. In regard to prevention of spread of infection, isolation of infected patients and elimination of infective attendants must be practised. The routine wearing of a gauze mask, practised at Guy's Hospital, should be seriously considered.

[1] *Guy's Hospital Reports*, July, 1930.

Abstracts from Current Medical Literature.

THERAPEUTICS.

Cerebro-Spinal Syphilis.

E. C. Menzies (*Canadian Medical Association Journal*, November, 1929) discusses the treatment of cerebro-spinal syphilis. Forty patients suffering from general paralysis of the insane were treated with malaria inoculation. Under this treatment sixteen general paretics recovered sufficiently to return to work for periods of nineteen months to three years and eleven months, and are still at work. "Tryparsamide" was used in forty-one similar cases prior to the use of malaria and nine of the patients are still at work. The results with malaria were obviously much better than those with "Tryparsamide" and it is now the custom to treat general paretics with malaria followed by "Tryparsamide" for twelve to fifteen weeks. Eighteen patients with other types of cerebro-spinal syphilis were treated in the same way. In two instances the malaria hastened death, but fifteen patients showed definite improvement both clinically and serologically. As the outcome is uncertain in all patients with cerebro-spinal syphilis, it is held that these results are better than those usually obtained with other methods of treatment. As the patients mentioned herein had all undergone prolonged treatment with "Salvarsan" and mercury prior to coming under observation, it was agreed that these drugs had been of small value, and they have been discarded by the author in the treatment of general paralysis. In weakly patients over fifty years of age malaria treatment was withheld while an attempt was made to build them up with "Tryparsamide" and general measures, but this failed and the author concludes that malarial treatment should be administered in all cases, as it alone holds out any chance of success. No difficulty was found in controlling the malaria; for this purpose 0·45 gramme (seven and a half grains) of quinine-urea-hydrochloride was given intravenously, followed by 0·6 gramme (ten grains) of quinine sulphate given thrice daily for three days and 0·3 gramme (five grains) thrice daily for the next four days.

The Use of Phenobarbital.

J. H. Huddleson (*The Journal of the American Medical Association*, November 23, 1929) reviews two series of patients treated by means of phenobarbital. The patients in the first series numbered one thousand and were not under such close observation as those numbering one hundred and forty-seven in the second series. The latter were also exposed to effective doses of the drug for much longer periods. Epigastric pain was complained of by one patient in the first series and seven in the second. The daily dose of phenobarbital in the patients showing this symptom was generally well below 0·18 gramme (three grains) and never exceeded 0·25 gramme (four and a quarter grains).

Therapy in Hæmophilia.

W. W. Payne and R. E. Steen (*Irish Journal of Medical Science*, November, 1929) discuss hæmostatic therapy in hæmophilia. The bleeding time may be normal in hæmophilia. A simple needle puncture in a normal subject often stops bleeding before clotting can have occurred; this may be due to contraction of capillary walls or aggregation of platelets sealing the injured wall. In hæmophilia there is no damage to the capillary wall and the platelet count is normal, hence a normal bleeding time is often found. The prolonged bleeding times sometimes found are probably due to puncture of larger vessels and in these circumstances formation of a clot is necessary to supplement the above two factors. In thrombocytopenic purpura capillaries are easily damaged and the platelet count is low and so, despite a normal coagulation time, bleeding time is definitely prolonged. In five cases of hæmophilia the bleeding time was found to vary between fifteen seconds and fifteen minutes, so that it is of no value in indicating the liability to hæmorrhage in this condition. The coagulation time is the important factor in hæmophilia. The normal time being one minute forty-five seconds, by the Dale-Laidlaw method, below one minute thirty-five seconds or over two minutes is abnormal. In the five cases reviewed coagulation time was estimated before treatment and at quarter hourly to hourly intervals afterwards. The substances used in treatment were given in a single dose and later in a series of doses daily for several days. The following methods were employed: Hæmoplastin given subcutaneously, "Coagulin Ciba" given orally and subcutaneously, fibrogen (Merrell) given orally and subcutaneously, protein shock, calcium lactate given orally, calcium chloride given intravenously, horse serum given subcutaneously, citrated human plasma given intramuscularly, intraperitoneally and intravenously, citrated human blood given intravenously, and liver treatment. Apart from citrated whole blood and citrated plasma given intravenously, none of the substances mentioned above had any but a transient, unimportant effect. With protein shock treatment and with calcium chloride a transient hypercoagulability was produced followed quickly by a condition of hypocoagulability. Intravenous citrated whole blood or plasma was found most useful both for treatment and for preoperative prophylaxis.

Treatment of Syphilis.

J. H. Stokes (*The Journal of the American Medical Association*, April 5, 1930) discusses treatment problems in syphilology. Thirty or forty injections of a reputable arsenical preparation should be given in three courses of ten or four courses of eight injections. No complete rest from treatment should be allowed for at least one year after the last symptom and sign have disappeared, the patient being kept on arsphenamine, bismuth or mercury by inunction or injection. Treatment should never be stopped or materially altered within a year of the first failure to obtain a response to the Wassermann test. A complete spinal fluid examination should be made in the first six to twelve months. The above treatment should be carried out when syphilis is diagnosed by any method of diagnosis. The sexual partner should be mechanically protected from the infected mate for five years from the time he acquired the disease, and every syphilitic woman should be treated through every pregnancy. The value of bismuth in treatment is yet undecided and it is still wise to give at least one-third of the course in mercury by injection or inunction. Bismuth should never be substituted for arsenical preparations in early stages and the main reliance should be placed on arsenicals, but arsenicals should not be used alone. Bismuth may relieve the lightning pains of tabes and it is useful in beginning the treatment in late syphilis, but it may do harm in late cardio-vascular and hepatic syphilis. If in doubt a soluble mercurial salt, such as the succinimide, is most reliable. Intravenous use of bismuth is contraindicated. A good bismuth preparation such as the fat-suspended potassium bismuth tartrate, the salicylate or the glucose-suspended hydroxide or metallic bismuth should be used. In patients who react strongly to soluble bismuth salts such as sodium bismuth tartrate, an insoluble salt such as quinine bismuth iodide in fat suspension should be combined. Arsenical preparations should not be used when the myocardium is injured, as determined by coronary symptoms and the electrocardiogram. In neurosyphilis the necessity of institutional care for malarial treatment limits its scope and suggests that "Tryparsamide" should be tried first, although it sometimes injures vision and may set up acute nervous symptoms in wrongly selected patients. Intraspinal treatment is being less used. In every case a careful examination and consideration of the activity of the disease are very necessary before treatment is commenced and a small initial dose should be given in any doubtful case. No initial dose should be more than half the full dose. In robust patients with early syphilis a small initial dose of arsphenamine may be given, but in all patients in the later stages of the disease when the heart, the nervous system, a viscus or a special sense organ is affected or suspected, injections of mercuric succinimide eleven to sixteen milligrammes (one-sixth to one-quarter of a grain) into the buttock should be given three to

five times a week, up to twenty injections, or from four to twelve weeks of inunction. Alternatively, an insoluble bismuth salt for six to twelve weeks may be given intramuscularly prior to the use of injections of an arsenical preparation. Mercury and bismuth are used as above in preparation for the arsenical drug in order to avoid the accidents due to lighting up a focus of syphilis. Such preparation is especially needed in syphilitic conditions of the eye, the cardio-vascular and the nervous systems. Arsphenamine requires expert preparation; "Neoarsphenamine" is more convenient. "Sulpharsphenamine" is contraindicated on account of greater toxicity. Injection should be made very slowly. Ephedrine in fifty milligramme doses is useful for prevention and treatment of arsenical reactions.

NEUROLOGY.

Amusia (with Aphasia) Affecting a Professional Pianist.

A. Souques and H. Baruk (*Revue Neurologique*, April, 1930) record a case which is interesting because it affords the rare opportunity of studying the effect of aphasia upon the capacity for music in a professional pianist. The aphasia was of the Wernicke type, total and complete, and although examination was conducted under difficulties, it was demonstrated that notwithstanding the high degree of speech loss, the capacity for music was relatively well preserved. The patient played spontaneously, from memory and almost faultlessly, a long piece of dance music. Being wholly "word deaf" it was impossible to get her to understand requests to play other pieces from memory. It may have been that she only had the one piece memorized; at any rate she played no other. By sign language, however, she was induced to play all the scales and did so with facility. It was difficult to judge how far her ear for music was affected; it could only be said that when familiar airs, such as the "Marsei"aise," were played to her, they were vaguely received. This much, however, was sure, the striking of a discord was instantly and painfully appreciated. Her ability to read music was remarkably intact. Thus, notwithstanding that she was unable to decipher one word of printed language or even recognize the letters of the alphabet ("word blindness"), she correctly read and played a series of pieces of music set before her. She also gave proof that she could "solfa" the diatonic scale correctly. In copying music she placed the notes accurately on the staves. The autopsy showed an old softening in the left hemisphere involving the whole zone of Wernicke (the white substance and gyri surrounding the extremities of the Sylvian and first temporal fissures). A series of complete horizontal sections of the hemisphere illustrated the lesion. After quoting corresponding cases of aphasia with relative integrity of musical capacity, the author considers the question whether a "centre" for music independent of that for language exists. The conclusion is that, great as the probability of this may be, the question cannot be answered until cases of the converse condition, pure amusia without aphasia, are proved by autopsy.

Spinal Symptoms in Chloroma and Leuchæmia.

Macdonald Critchley and J. G. Greenfield (*Brain*, April, 1930) record four instances followed by autopsy of the association of spinal symptoms in chloroma and leuchæmia which, though seldom mentioned, is actually not uncommon. Perhaps the severity of the illness due to the leuchæmia or chloroma sometimes masks or leads to overlooking of the nervous symptoms. Early indications are subjective complaint of pain, referred to the back or the legs, suggesting sciatica, and a feeling of stiffness in these parts. In the course of some days, or it may be weeks, this is followed, perhaps abruptly, by complete paraplegia. Objective sensory loss, sphincter disturbance and bedsores are usual accompaniments. The cerebro-spinal fluid has been rarely examined, but in one instance (Heissen) the finding of myelocytes or myeloblasts therein was diagnostic of myelogenous leuchæmic paraplegia. The commonest anatomical change is an infiltration of the spinal meninges and roots with leucocytic masses which may encircle the cord, like the rind of bacon. Such infiltration induces secondary anæmic and ischæmic changes in the spinal substance which account for the paraplegia.

Crural Paralysis in Herpes Zoster.

Lucien Cornil (*Revue Neurologique*, February, 1930) records an example of the somewhat rare complication of *herpes zoster* with crural paralysis. The patient was a female, aged fifty-eight years, who was attacked by a painful and typical herpetic rash over the front of the left thigh in the distribution of the second and third lumbar nerve roots. Fifteen days thereafter an unmistakable paralysis of the left quadriceps muscle appeared. A decided degree of atrophy with a partial reaction of degeneration ensued and not until six months had passed did recovery take place. In discussing the pathogeny of this syndrome reference is made to that inflammation of the geniculate ganglion which has produced herpes of the auricle along with facial paralysis (Ramsay Hunt), and to recorded instances of cervical and brachial herpes attended by paralysis (Souques and others). It is pointed out that attempt has been made to explain these by assuming that the well known "posterior root ganglionitis" of *herpes zoster* may break through the capsule of the ganglion and involve the anterior root, an event which confinement within a bony canal might assist. But Cornil rejects this mechanical hypothesis, at any rate for paralyses of the leg, indicating for one thing that the lumbar ganglia are not confined within intervertebral canals. He prefers an explanation recently offered by Lhermitte and Nicholas, that in exceptional cases of *herpes zoster* the characteristic inflammatory reaction attacks not only the posterior root ganglion, but the anterior root and the corresponding anterior cornu of the spinal cord.

The Control of Convulsive Seizures by Dehydration.

Temple Fay (*Journal of Nervous and Mental Diseases*, May, 1930) assumes that the convulsive state, whether acute or chronic, is associated with increased amounts of supracortical subarachnoid fluid. His chief reasons are: That such excess of fluid has been seen at operation and autopsy in the acute and chronic epilepsies; that the same has been amply confirmed by encephalography; that convulsive states have been induced in animals by "water intoxication"; and that the subarachnoid villi and Pacchionian bodies undergo morbid changes in epilepsy. The emerging suggestion is that this state of excessive hydration disposes the epileptic to seizures and that the maintenance of a satisfactory balance of fluids by continued dehydration is a rational form of treatment, which, though it may not cure, may control, just as other measures may control diabetes. The paper is a long one; it contains an elaborate theoretical discussion which is passed over in order that the following practical points in treatment may be mentioned. The patient is first subjected to preliminary observation for a week or more to determine his average consumption of fluid relative to elimination. He is next submitted to encephalography, the special purpose of which is to map out the subarachnoid spaces. This entails complete cerebral dehydration and occasions intense headache, but in a series of one hundred epileptics has not been attended by untoward result. Then begins the restriction of fluid intake. This is an arbitrary figure, but the amount should never be less than 180 cubic centimetres (six ounces) nor more than 600 cubic centimetres (twenty ounces) per twenty-four hours, and at the same time a diet is chosen in which excess of water is eliminated, for example, toast instead of bread, and a minimum of watery vegetables. Laxatives in the form of salts help. Complaint of thirst usually does not persist for long and eventually with the cooperation of the patient a record showing an intake and an output of 4·5 litres (one hundred and fifty ounces) per week may be obtained and carried on for an indefinite period.

Special Articles on Diagnosis.

Contributed by Request.

XIV.

CHOLECYSTITIS.

Pathology and Ætiology.

CHOLECYSTITIS may be divided into acute and chronic varieties, though the division is by no means hard and fast, since acute cholecystitis frequently is superimposed on a chronic condition.

Acute cholecystitis may be catarrhal, suppurative or phlegmonous. The two latter conditions are dangerous to life and require urgent surgical treatment, but it is difficult to separate the acute varieties from one another clinically with certainty, though the general symptoms are usually more severe in the suppurative and phlegmonous conditions.

Cholecystitis is almost invariably associated with infection. Conflicting views are held as to the mode of infection and the nature of the causative organisms. Streptococci, staphylococci, *Bacillus coli*, *Bacillus typhosus* and pneumococci have been reported by different investigators. Apart from those cases of cholecystitis which follow an illness such as enteric fever or pneumonia, an indication of the source of the infection is usually wanting. A careful history may, however, throw some light on the mode of origin of the disease.

Wilkie's researches indicate that a streptococcus is most frequently found, especially in the wall of the gall bladder and in the cystic lymph gland, through which the lymph from the gall bladder drains. He states that the bile itself is sterile in the majority of cases.

Gall stones are usually associated with infection of the gall bladder, and it is now thought that the pure cholesterin stone, the multiple faceted stones composed of cholesterin and bilirubin and the calcium carbonate stones which are opaque to X rays, are in most cases infective in origin. The condition in which the mucosa of the gall bladder presents a "strawberry" appearance is due to the deposition of cholesterol esters in the cells of the mucosa and underlying stroma. Cholecystitis may occur at any age in either sex, but it is most often found in obese persons of sedentary habit after forty years of age.

Hurst considers that cholecystitis is the commonest of all chronic abdominal complaints.

Symptoms.

The symptoms of cholecystitis are as a rule gradual in onset. Flatulence during or soon after meals, a sense of fullness or oppression, sudden nausea, associated with aching under the right costal margin or in the right scapular region are frequent symptoms. Often a feeling of chilliness and an aggravation of these complaints are accompanied by an icteric or muddy tinge of the skin. Slight fever may occur, vomiting is not infrequent and the patient avoids potatoes and fatty foods, as they are found to aggravate his condition. Sometimes the flatulence is delayed till an hour or so after meals and may be accompanied by heartburn, waterbrash and epigastric pain. At times the symptoms resemble those of duodenal ulcer, except that they are not so readily relieved by food and not so regularly recurring.

Attacks of gall bladder colic are usually due to stone, though similar pains may occur with cholecystitis alone. The pains of colic are felt in the right hypochondrium or epigastrium, they sometimes pass through to the back in the region of the angle of the right scapula; they may last from a few minutes up to several hours. A careful history should disclose the severity of the pains which are such as to necessitate the cessation of any occupation for the time and may be agonizing, even causing the patient to roll about on the floor at times.

Acute obstructive cholecystitis may occur at any time; it is associated with acute pain, vomiting, furred tongue, fever and often shivering, with extreme tenderness and rigidity of the muscles in the right hypochondrium and a catch in the breath when this region is palpated during a deep inspiration; often the tense gall bladder can be felt when the rigidity of the abdominal muscles in this area relaxes. The symptoms of acute obstructive cholecystitis usually subside after a day or two, but a careful watch should be maintained for any indications of perforation or suppuration.

During an attack of acute obstructive cholecystitis at times a flat percussion note and crepitations may be detected at the base of the right lung, due to immobilization of the right cupola of the diaphragm. The most valuable sign of cholecystitis is the localized tenderness just beneath the ribs in the right hypochondrium.

In making an examination, the right hand is laid flat on the right hypochondrium with the fingers pointing upwards and towards the mid-line, the patient is encouraged to relax the abdomen and take a long breath, at the same time pressure is exerted backwards and upwards with the palpating hand; a sudden arrest of inspiration accompanied by an appearance of acute discomfort is very suggestive of cholecystitis.

Diagnosis.

The differential diagnosis is important.

Acute appendicitis can usually, but not always, be excluded by a careful history and by the lower site of tenderness.

Pyelitis and renal colic are more frequently associated with pain and tenderness in the right loin posteriorly as well as anteriorly and the pain may radiate towards the groin. The urine should be carefully examined for pus cells, blood and organisms; an X ray examination of the renal tracts may be necessary to exclude renal calculus. A movable kidney may give rise to doubt. Riedel's lobe of the liver at times causes similar symptoms and should always be borne in mind. Pneumonia or pleurisy at the base of the right lung is usually distinguished by a higher temperature and more rapid and restrained breathing. The sudden onset of heart failure, with enlargement of the liver and without œdema of the legs, occasionally is a real difficulty.

The symptoms of cholecystitis may simulate those of duodenal ulcer and a barium meal may be needed to decide the question. The neurotic patient is perhaps the most difficult of all in whom to exclude cholecystitis, but repeated examination will generally reveal a change in the symptoms and in the site of pain and tenderness. Duodenal drainage is a method of examination the value of which is variously estimated. A tube is passed into the duodenum and eight to sixteen cubic centimetres (two to four drachms) of 25% solution of magnesium sulphate are introduced. This is followed by a copious flow of bile which is aspirated and studied macroscopically and microscopically. This test is scarcely applicable by the general practitioner and there is no general agreement as to the significance of the results. Radiation of pain to the angle of the scapula and tenderness at this site or to the right of the eighth to eleventh dorsal spines is not infrequently found in cholecystitis. Leucocytosis may occur and may be an aid in excluding non-infective causes of pain in the right hypochondrium.

An important method of investigation in suspected cholecystitis is by X ray examination which may show shadows of gall stones in the right hypochondrium. If this examination is negative, a further test should be made as follows:

Sodium tetraiodophenolphthalein is given either intravenously or by mouth, the latter being now the method of choice. A dose of castor oil is given on Monday evening, an ordinary meal without meat is taken at 6 p.m. on Tuesday, then, starting at 8 p.m. on Tuesday, two capsules, each containing 0·32 gramme (five grains) of the drug are taken with water every fifteen minutes till twelve capsules have been ingested. The patient drinks a glass of water before going to sleep and lies on the right side in the early part of the night. If nausea is felt, a little sodium bicarbonate in water should be drunk. The capsules must not be broken. No aperient is to be taken, except the castor oil. Nothing to eat or drink is

taken on Wednesday morning, on which day the X ray examination is made at 9.30 a.m.

Capsules coated with keratin to prevent gastric digestion are made by various firms, "Tetragnost," "Kerasol" *et cetera*, also powders are available, "Shadocol," "Keraphen" *et cetera*, which can be dissolved in fruit juice and swallowed at a dose. Rarely patients complain of nausea or diarrhœa after ingesting the test dose.

The sodium salt is excreted into the gall bladder in healthy persons, the iodine being the opaque medium.

Several X ray films of the gall bladder region are taken at 9.30 a.m. and the patient fasts till noon, when more are taken. The patient then has lunch, consisting of milk or cocoa, bread and butter or scones, and the gall bladder is examined again radiologically about 2.30 p.m. The 9.30 and noon films should show a good shadow of the gall bladder, while a normal gall bladder should be empty at 2.30 p.m., as the ingestion of food, especially fats, causes the gall bladder to contract and empty itself normally in less than half an hour. If the gall bladder does not fill at any time and the dye can be seen in the films distributed through the colon, one is safe in stating that gall bladder disease exists. Failure to fill with dye means that the cystic duct is blocked, that the gall bladder is full of stones or bile, or that a diseased condition of the gall bladder wall is present and is interfering with the concentration of bile. I am indebted to Dr. J. G. Edwards for details about the X ray examination in these patients.

The diagnosis of acute cholecystitis is often easy enough, but at times one of the above conditions may give rise to grave doubt and it may be necessary to examine the patient at frequent intervals and to observe closely the pulse, temperature, respirations and lung signs, as well as the abdominal condition. A history of previous gall bladder colic or jaundice may decide the diagnosis.

Chronic cholecystitis may be more difficult to determine and observation over some period may be necessary before a diagnosis can be arrived at. Indigestion which does not respond to treatment with diet, alkalis and measures to relieve constipation, should always raise a suspicion of cholecystitis; but in the absence of a history of jaundice, gall bladder colic, palpable gall bladder or marked tenderness over the gall bladder, a definite diagnosis of cholecystitis should not be made as a rule, even though the X ray examination shows that the gall bladder fails to fill with dye. The history and the physical signs found by the physician should be mainly relied on.

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Honorary Assistant Physician, Sydney Hospital.

British Medical Association News.

SCIENTIFIC.

A meeting of the Victorian Branch of the British Medical Association was held at the Women's Hospital, Carlton, Victoria, on July 16, 1930. The meeting took the form of a series of clinical demonstrations by members of the honorary staff.

Epithelioma of the Cervix.

Dr. Edward White showed a single woman who had first been admitted four years previously with a large, smooth, firm tumour protruding through the cervix. She was single, aged forty and complained of menorrhagia. The condition had proved to be an inversion of the uterus and had been dealt with surgically by subtotal hysterectomy.

Three months before the meeting she had reappeared and had complained of vaginal bleeding for the previous six months. A large, soft, irregular tumour had been found again protruding from the cervix and filling the vaginal cavity. This had been an epithelioma of the cervix and had been treated by radium puncture. Eighteen needles containing forty-two milligrammes of radium had been inserted for seven days, or 7,056 milligramme hours. Now the tumour had completely disappeared. She had suffered from a rare condition of inversion of the uterus, treated by subtotal hysterectomy, and four years later had developed an epithelioma in the cervical stump.

Pregnancy in a Bicornuate Uterus.

Dr. White's second patient had five children. Four weeks previously she had had an early incomplete abortion with bleeding, for which curettage was done, but the hæmorrhage had continued. When seen by Dr. White the patient had been blanched and there had been vaginal bleeding. A small well involuted uterus had been felt to the left and a large, soft, smooth mass had filled the right fornix. A diagnosis of ectopic pregnancy had been made, but under an anæsthetic a sound had been passed up to the right into the soft tumour; on withdrawal the sound had then been passed up to the left into the "uterus." The uterus had been a bicornuate one, with a recent pregnancy on the right side. After dilatation of the cervix blood clot and decidua had been curetted away. Apparently the left cornua had been curetted in vain a month previously.

Dysmenorrhœa and a Bicornuate Uterus.

The next patient shown by Dr. White was aged twenty-one. She had been married two years and was sterile. She had complained of severe and intractable dysmenorrhœa for years, with no relief from any kind of treatment. On examination a small uterus had been felt to the right and running to the left transversely there had been what appeared to be a long, firm, thickened Fallopian tube. But the patient had asserted that the left tube and ovary had been removed by operation five years ago. A bicornuate uterus had been suspected and under an anæsthetic a uterine sound had been passed into right and left cornua. After dilatation of the cervix a small tube of radium had been inserted into the right cornua, in order to cause a cessation of the periods for one to two years and thus relieve her dysmenorrhœa.

Bacilluria.

Dr. White's fourth patient was a single woman, aged thirty-six, who had been admitted ten days previously with severe abdominal pain and tenderness below McBurney's point and with frequent and painful micturition. The urine had been loaded with *Bacillus coli communis*, blood and pus and the diagnosis of acute pyelitis had been made. For a week the usual treatment had been given, including large oral doses of potassium citrate, but without relief. Then ten cubic centimetres of 1% acriflavine solution had been injected intravenously and repeated three days later. The change had been dramatic; the temperature had become normal, the urine had cleared and had become free of organisms on culture, and the patient had been completely relieved of her uncomfortable symptoms.

Puerperal Sepsis.

Dr. White then displayed the temperature charts of several cases of puerperal sepsis.

The first patient had had several positive blood cultures growing hæmolytic streptococci. First a femoral thrombosis had developed and later sceptic bronchopneumonia and pleurisy with effusion, from which hæmolytic streptococci were recovered. Apart from good nursing which again was emphasized as being the most important part of the treatment, blood transfusions had been given on two occasions, each containing 568 cubic centimetres (twenty ounces) of citrated blood. "Sulpharsenol" had been given subcutaneously every alternate day for six doses. This patient was now recovering.

Four other patients with puerperal sepsis had recently been admitted from a private hospital. In three of these cases hæmolytic streptococci were obtained from the cavity of the uterus by intrauterine swabs. The discharge had been rapidly cleared up by Remington Hobbs's glycerine therapy, by syringing out the uterine cavity with glycerine. Three of these patients were convalescent, but

the fourth had developed septicæmia, with hæmolytic streptococci grown from blood culture. The condition was further gravely complicated by an extensive septic bronchopneumonia.

Adeno-Carcinoma of the Labium Minus.

DR. ARTHUR SHERWIN showed a patient, aged sixty-six years, who had a pedunculated tumour the size of a golf ball, attached to the left *labium minus* in the neighbourhood of the clitoris. Microscopical examination of the tumour after excision had revealed adeno-carcinomatous structure of peculiar type, simulating an epithelial tumour of the salivary glands. The patient was shown with radium emanation applied round the site of the growth and inguinal regions.

Carcinoma of the Colon.

Dr. Sherwin's second patient was a woman, aged fifty-five years. An abdominal section had been performed four weeks previously for acute intestinal obstruction and general peritonitis. A suprapubic drain had been inserted and colostomy of the sigmoid colon had been performed. Later the abdomen had been opened again and portion of the colon with an annular carcinoma had been resected and end-to-end anastomosis performed. The colostomy opening had not been touched and the pelvis had been drained. Some days later the bowels had acted normally.

Ventral Hernia.

Dr. Sherwin also showed a patient with a large ventral hernia. The patient had recently been delivered of a child. On her admission the pregnant uterus had prolapsed through the hernial opening.

Pelvic Drainage.

Dr. Sherwin also showed several patients to demonstrate three types of pelvic drainage.

One patient, aged twenty-two years, had undergone posterior colpotomy. She had been confined three weeks prior to the operation. The indication for operation had been a fluctuating mass in the pouch of Douglas.

Another patient had been submitted to extraperitoneal drainage through an incision parallel with Poupart's ligament for an abscess occurring five weeks after confinement.

A third patient, aged twenty-four years, had had double tubo-ovarian abscesses removed and later loculi of pus had formed in the pelvis. Suprapubic drainage had been established.

Pyelitis.

DR. J. LEON JONA showed skiagrams and charts illustrating the value of pyelography and pyeloscopy in determining structural and functional changes in the kidney pelvis and ureter as causes of pyelitis. Indications for treatment were suggested and actions of drugs illustrated. The use of "Uroselectan" was also demonstrated.

Pregnancy Following Radium Treatment for Menorrhagia.

DR. W. D. SALTAU showed a patient, aged thirty-eight years, who had first reported at the antenatal department when thirty-two weeks pregnant. She was a *multipara* and it was her seventh pregnancy. Eighteen months previously she had been a patient in the gynæcological ward and, subsequent to a diagnostic curettage for a menorrhagia, she had had radium inserted against the cervix. She had been given a dose of 2,400 milligramme hours. This had induced amenorrhœa for about six months, after which she had menstruated regularly for four months and had then become pregnant.

She was extremely stout and when she first reported at the antenatal clinic it had been difficult to palpate any presenting part. The vault of the vagina and cervix were fibrous and the tip of the finger only could be inserted into what felt like an external *os*. An X ray examination showed the fœtus to be lying transversely. Under a general anæsthetic external version had been performed, but a few days later the malpresentation had recurred. At the time of showing at the meeting she was full term.

The points of interest were: (i) The fact that she became pregnant after treatment with a fairly large dose of radium, (ii) the possible course of labour on account of the fibrous cervix, and (iii) the possible effect of the radium on the child, as it had been maintained that the subsequent mentality of the child was likely to suffer.[1]

Pregnancy Complicated by Pelvic Tumour.

Dr. Saltau's second patient was a *multipara*, aged thirty-nine, with the obstetrical history that seventeen years previously she had had her first baby which lived for six weeks. Subsequent to that she had had three miscarriages and she had one living child, born eight years before. There had been no pregnancy since then. She had reported at the antenatal clinic a fortnight previously with a history of three months' amenorrhœa. She had had a slight vaginal hæmorrhage a few days before reporting herself.

On examination the pelvis had been found to be filled with a tumour which was diagnosed as a retroverted gravid uterus. It had felt irregular, but vaginal examination had been difficult on account of much tenderness. She had been admitted for examination and replacement of the uterus under general anæsthesia and it had been found that she had a mobile tumour about the size of a three months' pregnancy, situated to the left of the uterus. The uterus had been replaced without difficulty. The provisional diagnosis was a pedunculated fibroid or an ovarian cyst. As she had only one living child, it was considered inadvisable to operate on account of the risk of abortion, and it was proposed to allow her to go to term, and if the tumour was likely to cause obstruction, to perform Cæsarean section.

Eclampsia.

PROFESSOR R. MARSHALL ALLAN showed a patient, aged thirty-five, who was recovering from *ante partum* and *post partum* eclampsia. There was no history of previous illness and the first labour four years previously had been normal. Preeclamptic symptoms—albuminuria and œdema—had been noted for a fortnight and had failed to react to treatment. The sudden onset of epigastric pain had been followed by two fits and shortly afterwards labour had commenced. The patient had been admitted to the Women's Hospital with the fœtal head born. Solid albuminuria, a blood pressure of 132 millimetres of mercury and only a moderate amount of œdema had been noted. Morphine 0·015 gramme (one-quarter of a grain) had been injected and irrigation of the lower bowel performed while ninety cubic centimetres (three fluid ounces) of paraldehyde had been left *in situ*. Two further fits had occurred. The patient had been semicomatose at first and had then become conscious and talked irrationally. She had complained of blindness and examination of the eyes had revealed swelling of both discs with hæmorrhages in one eye. General improvement had soon occurred and Professor Allan emphasized the importance of a water diet during the first three days of the puerperium. Plain water or 10% glucose in water should be given freely. Too early a return to milk might precipitate further eclamptic convulsions. He then discussed the future diet and the need to keep proteins down to a low level for a considerable period.

Chronic Nephritic Toxæmia.

Professor Allan also presented a patient, aged twenty-five years, who had a history of scarlet fever on two occasions, eight and three years previously. Her first pregnancy had terminated one year before with the birth of a premature fœtus of eight months. During that pregnancy she

[1] Two days subsequent to the meeting the patient came into labour. She was watched closely and after fairly strong pains for a few hours no dilatation of the cervix was taking place and the finger could not be passed through any internal *os*. It was then deemed advisable to perform Cæsarean section, the classical operation being done and sterilization carried out by resection of the tubes. A living female child weighing 4·38 kilograms (nine and three-quarter pounds) was delivered. Fortunately both abdominal and uterine incisions were made fairly high, as it was found that the lower uterine segment was shut off by dense fibrous adhesions between uterus and parietal peritoneum. The mother's convalescence was normal.

had been treated for albuminuria. Two months later she had been admitted to the Alfred Hospital and treated for postscarlatinal myocarditis. Biochemical tests had revealed a blood urea content of 35 milligrammes per 100 cubic centimetres and a urea concentration of 0·8%. The patient had been discharged in October, 1929, convalescent. She had been admitted to the Women's Hospital on July 5, 1930, with a history of sixteen weeks' amenorrhœa. Her general condition had been good and no signs of myocardial trouble had been observed. The blood pressure was 140 and had dropped to 120 millimetres of mercury with rest in bed. Renal efficiency tests had shown a blood urea content of 23 milligrammes and urea concentration of 1·3%. After rest in bed and diet for a week albumin had appeared in the urine and the blood pressure had risen to 140 millimetres of mercury. Further urea concentration tests had shown slight improvement to 1·7%. Professor Allan considered that as the clinical symptoms were not satisfactory after rest and treatment and the chemical tests were also too low, interruption of the pregnancy was indicated and he proposed to do this immediately. He emphasized the risks taken in persisting with pregnancy in cases of chronic nephritis when symptoms developed in the early months. The patient would be instructed as regards prevention of further pregnancy until such time as the renal tests showed marked improvement. In many cases this was not likely to occur.

Professor Allan also showed the chart of a patient, aged thirty-four, in her tenth pregnancy. There was a history of albuminuria with the first child, while the last four had been still-born. In every case premature labour had been induced because of toxæmic symptoms and eclampsia had developed with the last child. She had been admitted with a history of œdema of the legs and headaches of three weeks' duration, defective vision for three days and considerable diminution of urine for a similar period. The urine had been nearly solid on boiling and the systolic blood pressure 170 millimetres of mercury, with marked œdema of the legs and abdominal wall. The blood urea had been 16 milligrammes *per centum* and the urea concentration 0·9%. Starvation and free purgation had had little effect and after four days labour had been induced with a rectal tube, and a live infant weighing 4·38 kilograms (nine and three-quarter pounds) delivered. After delivery the albuminuria had rapidly disappeared and the blood pressure had dropped to 140 millimetres. The blood urea had risen to 24 milligrammes, while the urea concentration had decreased to 0·5%, an ominous sign as regards future pregnancy. Professor Allan demonstrated this chart as an example of the type in which further child-bearing should be prevented.

Professor Allan presented the notes of another case as an example of wrong interpretation of an important symptom. The patient had had six children, she was twenty-nine years of age and had a history of severe pre-eclamptic toxæmia in her previous pregnancy. She had been admitted with a history of thirty-two weeks' amenorrhœa and complaining of severe attacks of pain in the right upper part of the abdomen radiating to the right shoulder and accompanied by vomiting. She was rather obese and although nothing had been felt, pressure under the right costal margin had elicited tenderness. A provisional diagnosis of cholecystitis had been made by the resident medical officer. Further examination had revealed a blood pressure of 260 millimetres. The urine had been free from albumin and renal efficiency tests had revealed a blood urea of 35 milligrammes and urea concentration 2·5%. Examination of the fundi had shown albuminuric retinitis and hæmorrhages in both eyes. Induction of labour had been performed at once and a still-born fœtus delivered. Albumin had appeared in the urine during the puerperium and the blood pressure on discharge had been 180 millimetres of mercury. When she was seen three weeks later, it had been unaltered and the albuminuria had still been present.

Toxæmia of Unknown Origin.

Professor Allan presented the chart of a primipara, aged twenty-one, who had been three and a half months pregnant and had had the usual amount of nausea and vomiting. She had been improving, but according to her husband there had been some diminution in the amount of urine passed. Suddenly she had become comatose and had been sent into the Women's Hospital. On admission she had appeared to be a healthy woman and the physical examination had revealed almost no abnormality. The urine had contained no albumin, but had reduced Fehling's solution to a deep red when undiluted. After dilution the urine had not given this reaction. The blood pressure had been 130 millimetres of mercury. The patient had become conscious, but had seemed dazed and unable to answer questions. She had then become very restless and had rapidly passed into a state of acute mania. It had been decided to empty the uterus, and this had been done at one sitting. The general condition had shown no improvement and death had occurred within twelve hours. *Post mortem* examination had shown that the uterus was apparently normal, while the liver was slightly smaller than usual, yellow in appearance and macroscopically resembled one associated with toxæmia. There had been a certain amount of myocarditis, but the brain had been perfectly normal. Sections of the liver showed changes analogous to those seen in cases of delayed phosphorous poisoning.

NOMINATIONS AND ELECTIONS.

The undermentioned has been nominated for election as a member of the New South Wales Branch of the British Medical Association:

Lyon, Marjorie Jean, M.B., B.S., 1928 (Univ. Sydney), 104, Louisa Road, Long Nose Point, Balmain.

The undermentioned have been elected members of the New South Wales Branch of the British Medical Association:

Windeyer, Richard Michael, M.R.C.S., 1925 (England), L.R.C.P., 1925 (London), M.B., Ch.B., 1930 (Univ. Cambridge), 2A, Besborough Avenue, Bexley.

Sullivan, Arthur Bernard, M.R.C.S., 1928 (England), L.R.C.P., 1928 (London), 124, Ben Boyd Road, Neutral Bay.

Obituary.

ARTHUR NORRIS WILKINSON.

We regret to announce the death of Dr. Arthur Norris Wilkinson, which occurred at Bendigo, Victoria, on September 17, 1930.

The Armit Fund.

The following is a supplementary list of subscribers to the Armit Fund:

Dr. C. B. Blackburn, Dr. J. A. Bonnin, Dr. E. Brown, Dr. G. H. Burnell, Dr. A. R. Clayton, Dr. H. S. Covernton, Dr. R. W. Cowan, Dr. A. M. Cudmore, Dr. C. T. Ch. de Crespigny, Dr. C. F. Drew, Dr. M. Erichsen, Dr. T. G. Fleming, Dr. H. Gilbert, Dr. A. Giles, Dr. J. B. Gillen, Dr C. Gurner, Dr. H. Halloran, Dr. F. S. Hansman, Dr. F. R. Hone, Dr. F. S. Hone, Dr. K. Inglis, Dr. H. M. Jay, Dr. A. Britten Jones, Dr. E. Britten Jones, Dr. A. A. Lendon, Dr. H. A. McCoy, Dr. J. L. McKelvey, Dr. P. S. Messent, Dr. J. Morton, Sir H. Newland, Dr. F. St. J. Poole, Dr. W. Robertson, Dr. E. A. H. Russell, Dr. H. H. E. Russell, Dr. B. Smeaton, Dr. H. F. Shorney, Dr. G. Sprott, Dr. A. F. Stokes, Sir J. C. Verco, Dr. J. S. Verco, Dr. R. J. Verco, Dr. C. E. C. Wilson, Dr. T. G. Wilson.

Books Received.

THE ŒDEMA OF BRIGHT'S DISEASE, by Ch. Achard, translated by Maurice Marcus, M.B., B.S.; 1930. London: Kegan Paul, Trench, Trubner and Company Limited; Melbourne: W. Ramsay. Demy 8vo., pp. 242. Price: 9s. net.

DIATHERMY MEDICAL AND SURGICAL IN OTO-LARYNGOLOGY, by Dan McKenzie, M.D., F.R.C.S.E.; 1930. London: Kegan Paul, Trench, Trubner and Company Limited; Melbourne: W. Ramsay. Demy 8vo., pp. 198, with illustrations. Price: 10s. 6d. net.

THE MYCOSES OF THE SPLEEN, by Alexander George Gibson, M.D., F.R.C.P.; 1930. London: Kegan Paul, Trench, Trubner and Company Limited; Melbourne: W. Ramsay. Demy 8vo., pp. 182. Price: 12s. 6d. net.

Diary for the Month.

OCT. 7.—New South Wales Branch, B.M.A.: Council.
OCT. 7.—New South Wales Branch, B.M.A.: Organization and Science Committee.
OCT. 9.—New South Wales Branch, B.M.A.: Clinical Meeting.
OCT. 9.—Victorian Branch, B.M.A.: Council.
OCT. 10.—Queensland Branch, B.M.A.: Council.
OCT. 14.—New South Wales Branch, B.M.A.: Ethics Committee.
OCT. 14.—New South Wales Branch, B.M.A.: Post-Graduate Work Committee.
OCT. 21.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
OCT. 24.—Queensland Branch, B.M.A.: Council.
OCT. 28.—Queensland Branch, B.M.A.: Obstetrical Section.
OCT. 28.—New South Wales Branch, B.M.A.: Medical Politics Committee.
OCT. 30.—New South Wales Branch, B.M.A.: Branch.
OCT. 30.—South Australian Branch, B.M.A.: Branch.

Medical Appointments.

Dr. O. W. Frewin (B.M.A.) has been temporarily appointed Honorary Assistant Physician, "Mareeba" Babies' Hospital, South Australia.

* * *

Dr. H. McI. Birch (B.M.A.) has been appointed Acting Deputy Superintendent of the Parkside Mental Hospital, South Australia.

* * *

Dr. A. N. Magnus (B.M.A.) has been appointed by the Perth Road Board, Western Australia, to be Medical Officer of Health.

* * *

Dr. W. L. Colquhoun (B.M.A.) has been appointed Certifying Medical Practitioner at Ringwood, Victoria, pursuant to the provisions of the *Workers' Compensation Act*, 1928.

* * *

Dr. O. Joynt (B.M.A.) has been appointed Acting Superintendent of the Hospital for the Insane, Mont Park, Victoria, from September 8, 1930, pursuant to the provisions of the *Lunacy Act*, 1928.

* * *

Dr. V. P. Johnson (B.M.A.) has been appointed Acting Superintendent of the Hospital for the Insane and the Receiving House, Ballarat, Victoria, from September 11, 1930, pursuant to the provisions of the *Lunacy Act*, 1928.

* * *

Dr. W. De Witt Henty (B.M.A.) has been appointed Acting Superintendent of the Hospital for the Insane and the Receiving House, Royal Park, Victoria, from September 5, 1930, pursuant to the provisions of the *Lunacy Act*, 1928.

Medical Appointments Vacant, etc.

FOR announcements of medical appointments vacant, assistants, *locum tenentes*, sought, etc., see "Advertiser," page xviii.

CAIRNS HOSPITALS BOARD, QUEENSLAND: Medical Officer.
ROYAL NORTH SHORE HOSPITAL OF SYDNEY, NEW SOUTH WALES: Honorary Physiologist.
SYDNEY HOSPITAL, SYDNEY, NEW SOUTH WALES: Honorary Urological Surgeon, Honorary Clinical Assistant.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company, Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Members accepting appointments as medical officers of country hospitals in Queensland are advised to submit a copy of their agreement to the Council before signing. Brisbane United Friendly Society Institute. Mount Isa Hospital. Mount Isa Mines.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to "The Editor," THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

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